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AGARD REPORT No.758

Short Course on Cardiopulmonary Aspects of Aerospace Medicine

Addendum

NORTH ATLANTIC TREATY ORGANIZATION

ADVISORY GROUP FOR AEROSPACE RESEARCH AND DEVELOPMENT

(ORGANISATION DU TRAITE DE L'ATLANTIQUE NORD)

AGARD Report No.758 (Addendum)

SHORT COURSE ON CARDIOPULMONARY ASPECTS OF AEROSPACE MEDICINE

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PREFACE

In 1987, the AGARD Aerospace Medical Panel sponsored a Short Course on the Cardiopulmonary Aspects of Aerospace Medicine. The course was given at four locations: Fürstenfeldbruck, Federal Republic of Germany, 18—20 May, Athens, Greece, 21—23 May, Copenhagen, Denmark, 9—11 June and Eskişehir, Turkey from 15—17 June. The lectures and related case presentations were published and distributed prior to the course in AGARD Report No.758.

The lecture series was extremely well received. Based on the information received in the course critiques, over 85% of respondents rated the course content and faculty either 4 or 5 on a scale of 5.

At each location, the lectures provoked considerable discusssion. These discussions contained a great deal of valuable information, expanding and clarifying the material in the lectures, and giving Flight Surgeons from the host countries an opportunity to express their views, and to interpret the material in the perspective of the experience and priorities of their own Air Forces.

In the course critiques, over 85% of respondents indicated that a follow-up booklet containing summaries of the discussions would be valuable. This AGARD publication contains the transcription of the recordings of the discussion periods. Since the recordings were informal, speakers were not asked to identify themselves, and therefore may not be identified in the transcripts. At times, technical difficulties prevented transcription of the verbatim discussions; at such times, an interpreted version of the notes taken by the lecture staff has been included.

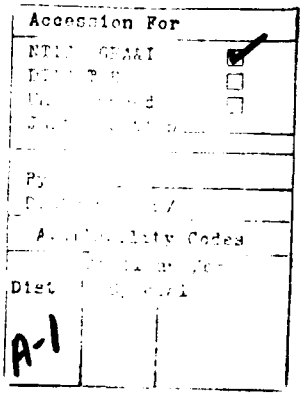
The essence of the course was summarized by the Course Director, Colonel Hickman, during his closing remarks in Copenhagen.

"Making decisions in aerospace medicine is very heavily dependent on defining who is 'normal'. In the USAF, in terms of making decisions about who is 'normal', we are rather dogmatic about the decision process. This is not because we are not scientists and are not sceptical by nature. It is because if you are going to have a decision rule you must have some way to operate, and you should not have to apologize for being arbitrary because you don't know who is 'normal' based on the currently available data. However, if you continue in that vein for ever, I think you have to be apologetic, and I think that you can only be dogmatic if you carry the flag that you will change the decision rules as the data allow, and that you will actively pursue the acquisition of new data.

"As you know, in our Air Force the decision rules are all pretty conservative about who is 'normal'. Proving who is 'normal' often takes observations of large numbers of people over time, and the accumulation of large denominators will be facilitated if NATO nations pool their data. Our decision rules are always becoming liberalized rather than made more narrow because we start off on a very conservative stance. There are no rules that will apply to all NATO Air Forces. Your decisions must depend on the size of your Air Force, the views that you have of the selection process, the data that you have available and the types of airplanes that you fly. These all make a big difference in the decision process.

"As to the question of who is 'normal', I believe that the NATO countries must join in and pool their data. It doesn't matter if we make different decisions from the data. I think that your denominators would add up so much more quickly if we could add our data together, especially in the area of selection. The US selection process is decentralized, and there is no hope of learning much from us about selection. As a matter of fact the reason we have so much to say about retention is because we are so poor at selection. I would urge those of you whose selection processes are centralized to write those data up and present it.

"I also think that it is important in every country where decisions are made about aerospace medicine, that the people who make the clinical decisions be given the opportunity to see aircraft accident investigations to develop a sensitivity about exactly what it is we are concerned about, because it is just not just an abstraction. There is a certain finality that comes home only after you have seen the exact results of an aircraft accident a number of times."



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PREFACE

En 1987, le Panel AGARD de Médecine Aérospatiale a organisé un Cours sur les aspects cardiopulmonaires de la médecine aérospatiale. Ce Cours a été présenté à Fürstenfeldbruck, RFA, du 18 au 20 mai, à Athènes, Grèce, du 21 au 23 mai, à Copenhague, Danemark, du 9 au 11 juin et à Eskişehir, Turquie, du 15 au 17 juin. Les exposés et les cas particuliers y afférant ont été publiés dans le rapport AGARD No.758 et distribués avant la présentation du Cours.

Les communication et les présentations des observations cliniques ont été très appréciées par les participants. Plus de 85% des personnes interrogées par questionnaire ont donné une note de 4 ou de 5 sur 5 en ce qui concerne le contenu et la pertinence du Cours.

Chaque Cours a été marqué par de longues discussions lors de la table ronde organisée en fin de séance. Une quantité non négligeable d'informations précieuses a été echangée lors de ces discussions, qui servaient à élargir ou à éclaircir les différents sujets traités par les orateurs. Ces informations donnaient aux médecins de l'Aéronautique des divers pays hôtes, la possibilité d'exprimer leurs opinions et d'illustrer les cours à la lumière de leur expérience personnelle, en fonction des priorités établies par leurs commandements respectifs.

Plus de 85% des personnes interrogées ont signalé l'intérêt d'une publication ultérieure, qui serait composée d'une série *de résumés des debats. Cette publication* AGARD contient la transcription des enregistrements faits lors des discussions. Ces séances étant d'un caractère non-officiel, il n'a pas été demandé aux participants de se présenter, et, par conséquent, les noms de certains participants manquent dans le texte. De temps à autre l'enregistrement a été interrompu à cause de problèmes techniques. Une version revisée des notes prises par les conférenciers a été incluse puor combler ces lacunes.

L'essentiel du Cours a été résumé par le Directeur du Cours, le Colonel Hickman dans son allocution de clôture à Copenhague, dont le text suite:

"La prise de décision en médecine aérospatiale dépend fortement de la manière dont on définit un sujet "normal". Dans l'USAF, nous avons tendance a être un peu doctrinaire en ce qui concerne cette définition. Nous sommes pourtant des scientifiques et de nature sceptique, mais dès que vous établirez des règles pour la prise de décision il vous faudra un "modus *operandi*" et on ne doit pas avoir à s'excuser même si nos décisions semblent arbitraires simplement parce que les données disponibles à l'heure actuelle ne nous permettent pas de définir ce qu'est un sujet "normal". Cela étant dit, si vous persistez dans cette voie, vous serez obligés de justifier vos actions et vous ne pourrez être dogmatique que si vous dites clairement que vous ne modifierez les règles de prise de décision qu'en fonction des données disponibles et que vous cherchez à acquérir de nouvelles données.

Comme vous le savez, dans l'USAF, les règles de prise de décision *concernant le sujet "normal" sont plutôt* conservatrices. La définition du sujet "normal" implique souvent l'observation de nombreux sujets sur des périodes relativement longues, et le collationnement de dénominateurs communs de plus en plus grands serait facilité si les pays membres de l'OTAN mettaient en commun leurs données. Nos règles de prise de décision évoluent toujours dans le sens d'une libération, puisque le point de départ est toujours conservateur. Il n'existe pas de règle applicable à toutes les armées de l'air des pays membres de l'OTAN. Les décisions que vous prenez dépendent forcément du nombre de personnes inscrites à votre tableau d'effectifs, de vos opinions concernant la procédure de sélection de ce personnel, les données disponibles et les différents types d'avion en service. Tous ces facteurs influents sur la prise de décision.

Quant à la question de savoir qui est "normal" je pense que tous les pays membres de l'OTAN devraient mettre leurs données en commun. Peu importe si les décisions que nous prenons ne correspondent pas . Je crois que nous pourrions collationner nos dénominateurs communs beaucoup plus vite si nous combinions nos données, surtout dans le domaine de la sélection. La procédure de sélection aux US est décentralisée et ce n'est pas nous qui allons vous apprendre grand-chose sur la sélection. En fait, si nous avons tant de choses à dire sur le maintien en condition du personnel, c'est parce que nous ne sommes pas très forts en sélection. Je conseille très fortement ceux d'entre vous qui utilisent des procédures de sélection centralisées de rassembler leurs données et de les présenter.

Je pense également qu'il est important, pour chaque pays où des décisions sont prises concernant la médecine aérospatiale, que les personnes responsables de ces décisions cliniques aient la possibilité d'assister aux enquêtes sur les accidents aériens, pour qu'elles soient sensibilisés au réalites de nos préoccupations, car nous sommes loin d'en faire abstraction. Il y a là une certaine finalité dont on ne se rend vraiment compte que lorsqu'on en a mesuré les consequences un certain nombre de fois lors des accidents aériens."

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by

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DISCUSSION - FURSTENFELDBRUCK

Question: If an applicant has PVCs on his screening ECG, is he disqualified from flying training?

L/Colonel Kruyer: If the only abnormality on the ECG is ectopy (PVC's), then they can be qualified either to get into flying training initially or to continue flying. They may be disqualified temporarily while a Holter monitor is assessed. If the Holter monitor discloses nothing other than PVC's, then a waiver is allowed. With an experienced aviator, if the Holter shows only rare or infrequent ectopy, they don't even require an evaluation at USAF/SAM. This is considered within normal limits and does not require a waiver.

Complexity probably should be disqualifying for entry into flying training until we know what truly represents normal findings and no aeromedical risk. Also, echocardiography should be done to exclude structural cardiac disease. At the young age of training applicants, coronary disease is unlikely but treadmill testing may be considered also for an arrhythmia evaluation.

Colonel Hickman: One thing that you want to know about with ventricular ectopy, is whether or not there is underlying structural cardiac disease, regardless of complexity or frequency. If you can't find any structural cardiac disease, then you have to look upon the ventricular ectopy as a normal variant. The rule ought to be, if there is no underlying organic disease, ventricular ectopy is considered a normal variant and is waiverable for someone who is already flying. However, normal variants often do not declare themselves except over time, and to take someone into flying training who has had demon
strated ventricular tachycardia, or who is in bigeminy 20 mins. out of every hour, even though you cannot find organic heart disease, would be a very poor bet.

Question: Last year, here at the Institute of Aviation Medicine at Furstenfeldbruck, during four or five hundred examinations, we had two hundred pilots who had ventricular ectopic beats, and roughly 30% had Lown grade 3 to 5. The question is what to do with these pilots? This is a dilemma for us. We put these pilots in the centrifuge, and until now we have a trend but we have no real decision. I would like to discuss this problem a little further. What about pilots who have salvos (with exercise), and in the centrifuge they don't have any salvos?

Colonel Hickman: We don't know for sure what to do with them either. We are stuck really with erring on the side of conservatism. In a clinical population, repeated salvos of three to four beats of ventricular tachycardia would't necessarily require medical treatment. They wouldn't be treated with anti-arrhythmic drugs unless there was underlying disease, e.g. coronary disease. On the other hand you are forced to worry about them in an aviator. Even a centrifuge run in which there is no ventricular tachycardia does not rule out the possibility that in actual flying missions where they are repeatedly pulling high levels of G, that it might not occur in that environment. So for the present time our policy is that if they have one episode of non-sustained ventricular tachycardia and their coronary angiograms are normal, we would consider that waiverable, based on our Study Group which showed that they are not at risk of sustained tachycardia or other events. Our aviators with normal coronary arteries and non-sustained monomorphic ventricular tachycardia of seven beats or less are returned to non-high performance flying. If they have another single episode one or two years later, when they are being retested, we do not disqualify them. However, if they have several episodes during the same evaluation, during treadmill testing for example, we consider that they are showing enough frequency to be at a higher risk of sustained ventricular tachycardia, and they are disqualified.

Question: What do you do with high degrees of ventricular complexity in combination with mitral leaflet prolapse?

L/Colonel Kruyer: For aeromedical purposes the two entities have to be considered as being causally related and this requires permanent grounding.

Question: What is the basis for your criteria of seven consecutive ventricular premature beats as being the limit for non-sustained ventricular tachycardia?

L/Colonel Kruyer: It is based on the fact that in our study group, all subjects had seven beats or less. It was a characteristic of the study group who had no structural cardiac disease that none of them had more than seven consecutive VPB's.

Colonel Hickman: We had never put anyone with ventricular tachycardia back on flying status until 1980. We were grounding them at the rate of one a month, and it was costing us a million dollars a month. In order to present a waiver argument for some cases of ventricular tachycardia to our Surgeon General, we undertook a very extensive retrospective study and looked for tests which would absolutely differentiate those with organic disease, including a full non-invasive work-up and catheterization. Seven beats was the limit of our experience. In order to present it to our Surgeon General and stay within our experience, we defined our limit as seven beats. In fact, I would rather see someone who had fifty beats because we then have some hemodynamic information which would be very helpful. Unfortunately, in our retrospective study of forty five aviators, none had more than seven beats.

L/Colonel Kruyer: The ventricular tachycardia study group started in 1978, and since then there have been thirty one people enrolled, and eighteen of them have been waivered. The others have been disqualified, mostly because they had underlying coronary disease. In 1981, the study group was enlarged to include ventricular tachycardia discovered on Holter monitoring as well as during exercise. The other criteria are the same, that is, seven beats or less. There have been twenty people under that grouping, and eight of them have been waivered. In the twenty-six of a total of fifty-one who have been waivered there have been no coronary artery disease events. They are annually evaluated at USAFSAM.

Question: Do you do electrophysiologic studies and ventricular stimulation as part of your work-up of ventricular tachycardia.

L/Colonel Kruyer: It is not done at the School of Aerospace Medicine. Rather than stimulating the right ventricle to try to induce ventricular tachycardia, we look for underlying cardiac disease. It would be difficult to interpret such data, and in addition, such studies are much more dangerous than electrophysiologic studies for bypass tracts, and we are reticent about doing that to asymptomatic individuals for occupational reasons. There is no data available on that in the standard cardiac literature.

Question: What do you do with ventricular extrasystoles during the stress test without ST changes?

L/Colonel Kruyer: We code those as significant stress arrhythmias, but if they don't have venricular tachycardia they do not reach a threshold for angiography. We rarely terminate treadmill in healthy aviators for complex ectopy unless they really have symptoms or unless they have a run of ventricular tachycardia that is quite lenghty.

Question: In those cases where they don't have a tachyarrhythmia, would you recommend a 2D echo or a thallium study?

L/Colonel Kruyer: I think if you have complex ventricular ectopy with a negative ST segement response, if the patient is an aviator, he should have a 2D/Doppler echo study and exercise thallium study. If the ectopy were high grade but with no salvos, he probably should also have an exercise MUGA to make sure that you are not dealing with an early cardiomyopathy.

DISCUSSION - AIRCREWS

(Discussions on this lecture were not taped)

Question: What is your disposition of an aircrew with WPW who manifests supraventricular tachycardia with exercise?

L/Colonel Kruyer: At USAFSAM, we would recommend disqualification from all flying duties for such an individual. I realize the decision in other NATO countries might be different. Under some circumstances, we would consider a waiver for a brief, self-limited episode of SVT, provided there was no bypass tract, no recurrence, no evidence of coronary disease, and no symptoms with the tachycardia. But with the anatomic substrate for a recurrence present in the form of a bypass tract, we would not consider a waiver.

Question: What is your approach to an aircrew applicant with a right bundle branch block?

L/Colonel Kruyer: The USAF at present would not accept such a candidate for flying training. We are at present considering a revision of this policy, if other non-invasive studies are normal. We are also considering a recommendation for local, rather than USAFSAM, evaluation for acquired RBBB in experienced aviators.

I should explain that the School of Aerospace Medicine at Brooks Air Force Base is a referral center for evaluation of trained USAF aviators, and is not used for candidate evaluation. The USAF does not have a central screening facility for aircrew candidates.

Colonel Hickman: The USAFSAM RBBB Study Group will have 20 years of data in 1988, with a mean follow-up of about 10 years. Based on that data, we will recommend that candidates with RBBB be accepted for aircrew training.

Question: You define recurrent superventricular tachycardia as two or more runs of 3 or more beats of SVT. In what time frame must these occur to be considered "recurrent"?

L/Colonel Kruyer: During the six month grounding period for work up of SVT, aviators have at least 3 Holters, as well as two exercise tests, one a standard treadmill test, and the other an exercise thallium study. If there is any repetion of SVT during the six month period, it is considered "recurrent".

Dr. Masdrakis: In aviators with left bundle branch block, how often do you repeat their coronary angios?

L/Colonel Kruyer: That is an excellent question - what is the duration of validity of angiograms in ensuring normal coronary arteries. We would likely want to repeat the angios every 10 years, providing the initial ones were normal, but only if there were changes in non-invasive tests, such as the stress thallium or MUGA scans, to suggest the development of coronary disease.

DISCUSSION - COPENHAGEN

Question: When is coronary angiography to be performed for ectopy?

L/Colonel Kruyer: Coronary angiography is not done for anything less than tachycardia. Frequent PVC's, pairing, or multiformity do not require angiography unless indicated for some other reason, for example an abnormal thallium or abnormal treadmill test. US Air Force aviators with ventricular tachycardia who meet other criteria for returning to flying duties must have coronary angiography before being given a waiver. Anyone with supraventricular tachycardia over the age of 35 must have coronary angiography, as well as those under the age of 35 with significant risk factors.

Question: For those given waiver for return to flying duties, how frequently are they re-evaluated and what does the re-evaluation involve.

L/Col Kruyer: Re-evaluations for ectopy are generally carried out at the School of Aerospace Medicine every 3 years. For aviators given a waiver to return to fly for 3 years, the waiver expires at the end of the 3 years and the aviator must return for re-evaluation. All the re-evaluations are standard non-invasive work-ups, involving history, physical and lab work, a resting ECG, Holter monitor, echocardiogram, treadmill and usually a thallium. Some of the re-evaluations include a MUGA or radionuclide angiogram at rest and exercise but that depends on what they are being followed for, for instance mitral regurgitation or aortic insufficiency.

Colonel Hickman: The Brooks evaluations do not supercede our local evaluations. The real reason that we do these repeat centralized evaluations is because most of the disorders that we are talking about were at one time non-waiverable in our Air Force, for example, before 1980 we had never returned anyone to flying status with ventricular tachycardia. Most physicians when they talk about ventricular tachycardia are dealing with their own experience in a clinical population where such findings are generally malignant and difficult to associate with flying duties. We finally went to our Surgeon General with enough retrospective data to say that the trend was favourable and with adequate diagnostic tests to identify a safe separation point. Our agreement with our Surgeon General is that he will allow us to put people back on flying status of some category contingent upon the fact that we can prove that this does not represent a disease diagnosable by current technology. We must prove on a recurrent basis that this is true, so really what we are doing is natural history studies.

L/Col Kruyer: Even aviators that have been disqualified from flying duties we invite to return to Brooks for follow-up studies for their own benefit, which also allows us to continue our data collection. More frequent follow-up may have to be done at a local level for certain conditions, for example a recurrent v. tach. One problem is that most of these conditions are asymptomatic and the individuals are reluctant to go to a local clinic facility where it may be difficult for them to get into and be seen by someone because to the clinician they don't have a problem.

Question: Have there been any in-flight incidents due to cardiac conditions?

Colonel Hickman: There have been some aircraft accidents where clearly the individual had coronary disease and the scenario, including the analysis of communication before the crash made it very definitely sound like a coronary event. There also have been a number of aircraft accidents where we just don't know what happened; that is somebody crashes without any communication back to tower or to other aircraft (which could be due to disorientation) but if there is significant coronary disease found at autopsy, the possibility of incapacitation has to be raised

Question:

(a) Is the USAF policy on frequency of ECG's, that is an initial ECG at 20, and then the next one being at age 35, primarily because of logistics, or for other reasons?

(b) Do you maintain your ECG repository on file, or have you a computer system for data?

L/Col Kruyer:

(a) It is true that in the USAF the number of ECG's would be large if done annually, but with the screening ECG at 20 and regular flight physicals on an annual basis, it's unlikely that anything new would turn up on an ECG before age 35 besides normal variants, and we would be spending a lot of physician's time investigating normal variants. Some of the NATO countries have an even more extensive initial screening including an echocardiogram and certainly in those circumstances you would be unlikely to uncover anything abnormal for at least a few years unless there was a clinical occurence. So it's primarily because the ECG is such a poor screening tool after an initial normal that we don't repeat it until age 35.

(b) The ECG's are all stored on microfilm. We can get 30 studies on one 4 inch by 5 inch microfiche. We are in the process of digitizing the library so that we can store everything as digitized signals. This will be more efficient and cost effective when it's completed. Also, when you want to make hard copies from microfilm, the copies are very poor, whereas with the digitized system, the reproduction will be as good as the original hard copy.

Colonel Hickman: We have spent millions of dollars over the last number of years on computerized systems for interpretation, but the blunt truth is virtually all of the computerized interpretation systems are fine for clinical problems, but most of our ECG's are on normal individuals and none of the systems are capable of comparing minute to minute, hour to hour, day to day, year to year changes in the T waves and ST segments. The reason we are digitizing is strictly for storage, not interpretations.

Question: In your description of left ventricular hypertrophy you mentioned your modified criteria which are changed in order not to catch a large number of normal people in your net. What are your criteria in other non-invasive studies, for instance in the echocardiogram, or the ejection fraction of the MUGA?

I think it's a very difficult problem to define normal values and abnormalities in such populations. How do you modify your normal values in such tests to avoid getting too many false positives?

L/Col Kruyer: As a general statement, we use whatever the criteria are in a clinical population until we have enough criteria in our own unique population to define the normals for that population. For instance, for left ventricular hypertrophy, we studied a large population of normals who had LVH by ECG criteria and found they did not have LVH with further study. This led to our redefining the normals for that population, which can only be applied to that particular population.

Colonel Hickman: Another answer to the question is that the various studies should not be looked on as being applied to populations with the same degree of background prevalance. These studies should be stratified and it's the stratification of who gets the test that is really important. There is no need to do tests in an across the board fashion. It's not a diagnostic problem and you can use the same criteria that you use in a clinical population once you decide the persons who should be tested.

The standard electrocardiogram is a highly non-specific tool, but once you have decided who needs further study based on the criteria that we use, for example for LVH, the echocardiogram becomes a highly specific tool. To apply any test in a regular across-the-board fashion though, is highly unrewarding. It's not a matter of setting standards for a large battery of tests that should be applied to everyone, but rather deciding which tests should be given to whom.

Question: Regarding the question of LVH and it's relationship to exercise, what do you do with individuals who have LVH on ECG and echocardiography, yet on questioning do not give a history of extensive exercise. Do you still require these individuals to refrain from all exercise for a period of six months and then retest them?

L/Col Kruyer: What's in the literature revolves around isometric exercise causing concentric LVH. Aerobic exercise tends to cause LV dilatation and maybe some proportional hypertrophy. However, we have seen pure concentric hypertrophy without dilatation in people who only run, and don't perform any weight training or other isometric exercise. Even in individuals who don't give a history of exercise, we do require a period of six months without any exercise and then retest them. The issue is whether it's a hypertrophic process or an athletic heart, and we will send such individuals home on no exercise and repeat their echo in six months. We have seen some pretty impressive 13 millireter wall thickness shrink down to 11 millimeters in a period of 3 to 6 months by having an individual stop running.

Question: How long does it take to build back up?

L/Col Kruyer: We really don't know. What we tend to do now is to ignore hypertrophy if it redevelops with exercise once we have demonstrated that it goes away with no exercise.

Question: Do we see LVH more commonly in high performance pilots than we do in non-high performance pilots?

L/Colonel Kruyer: We haven't specifically looked at that. All of our high performance pilots are advised to lift weights to improve their G tolerance so should it occur, it might be due to the weight training rather than pulling G.

Colonel Hickman: This is one of the purposes of the long term echocardiographic follow up study that is being organized by NATO/AGARD. The Aerospace Medical Panel is sponsoring a long term collaborative follow-up study to try to determine whether or not there are deleterious effects of long term +Gz exposure, comparing different coherts of people who fly different airplanes, age matched over time.

The French have reported right ventricular enlargement in Mirage pilots compared with a similar group of age-matched transport pilots.

Question: What is the USAF policy on left anterior and left posterior hemi-block?

L/Colonel Kruyer: The evaluation and disposition of aviators with left anterior and left posterior hemi-block is basically the same as for right bundle branch block. If we have a right bundle branch block that is complicated by hemi-block or so-called bifasicular block, then they may either be disqualifed or at best, they will require a more intensive screening protocol including coronary angiography and an electrophysiological study.

Colonel Hickman: We stopped doing coronary angiography and electrophysiological studies in 1978 for right bundle branch block after a study group involving several hundred aviators. At that time, we said that we would continue to do angiography and EPS for bifascicular block, but we have not had a case since. Right bundle branch block that we see as a serial change in aviators occurs very distally, beyond the moderator band. In such a population, we have just not found associated hemiblock.

Comment: We also studied an older population of civil aviation pilots in who we see right bundle branch block complicated by hemiblock, and they are still allowed to fly.

DISCUSSION - ESKISHER

(Based on Notes of Discussion Periods)

Question: Could you explain the USAF categorical system for granting waivers?

Colonel Hickman: Until recently, the U.S. Air Force had a "fly one-fly all" policy. Recently, a categorical system for medical waivers was introduced, but the commander of Strategic Air Command (SAC) and Military Airlift Command (MAC) made it quite clear the only way they would accept aviators with medical problems was if the problem was uniquely related to or would be aggravated by G forces. They insisted that even though their aircraft were multiplace, that each of the crew members, including the pilot and copilot, had a critical role to fulfill in at least certain aspects of the mission, and could be considered as redundant. And so, if we doctors are concerned that the aviator has a medical problem that might cause incapacitation, or jeopardize the successful completion of a mission, the SAC and MAC commanders don't want that aviator on their flight decks any more than the TAC commander in his fighter cockpit.

What is your policy here in Turkey regarding categorical waivers?

Answer: In Turkey, we have a "fly one/fly all policy".

NONINVASIVE METHODS FOR THE DETECTION OF CORONARY ARTERY DISEASE IN AVIATORS – A STRATIFIED BAYESIAN APPROACH

by

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DISCUSSION - FURSTENFELDBRUCK

Question: The total/HDL cholesterol ratio appears to be of value only when the total cholesterol is borderline. If the total cholesterol is greater than 300 mg/dl, such an individual appears to be at increased risk independent of the HDL. Could you comment.

Colonel Hickman: Our angiographic data do not bear that out. We are finally discovering why some individuals with a cholesterol of 180 (mg/dl) have serious coronary artery disease, and that is because they inheritated HDLs of 20 (mg/dl). We are also finding out how people with cholesterols of 300 can go for a life time and not have coronary disease, and that is because they were fortunate enough to inherit an HDL of 75 or 80. Most of the people who live to be over 80 in North America have very high HDL's. Until some new angiographic data comes along, I am still going to put a lot of credence on the ratio.

Question: Do you feel it is of any value in doing HDLs in persons who have a normal cholesterol say under 200?

Colonel Hickman: Yes. If you have a cholesterol of 200 and you are unlucky enough to have an HDL of 20 (mg/dl), and you are a young person, we think you are at a much higher risk of coronary disease, and that (such an) individual must be made aware of that information. In the future, we think that the apolipoproteins will provide us with a great deal more information, and will make the ratio itself obsolete.

Question: How do you manage aviators with a ratio above 6 and a high cholesterol?

Colonel Hickman: It is a fairly standard clinical approach. The first is to characterize the lipid abnormality, the second is a strict dietary approach coupled with achieving appropriate body weight for height, and the third is an exercise program after appropriate screening. If, in an aviator, these measures do not produce satisfactory results, we will go to cholestyramine, which is the only drug that we feel you could currently safely use in an aviator, to lower his lipids, because of its non-absorbed nature. Drugs like mevinacin and gemfibrazole may become acceptable in aviators after appropriate study, but currently they are not. That is basically how we would handle one of our aviators. We find that most of our aviators are not really familial hyperlipidemia, but rather they got their elevated lipids in the usual ways, that is through lifestyle.

Question: It is interesting that you are doing your study on detecting asymptomatic coronary artery disease only in TAC (Tactical Air Command). Is that because in multicrew cockpits the concern about coronary events is not as great in that there is a co-pilot take over in the case of an emergency?

Colonel Hickman: We started the study with TAC because we wanted a population in the continental U.S., and because our concerns about asymptomatic coronary disease were highest in that group. TAC commanders themselves were also very interested in the program.

Question: What is the cost per case to find asymptomatic coronary disease and is that approach cost effective in the case of two-pilot aircraft? Do you think such a study needs to be expanded Airforce wide?

Colonel Hickman: We believe such programs should be expanded airforce wide, but we want to make sure that it is the right program. We don't expect the number of caths to increase but rather to be doing them on the right people as a result of pre-selection screening. We could not give you an accurate current cost per case because the program is in a state of flux. We should have good cost data when the thresholds are finalized.

Question: What about the risk index in a general population other than aviators?

Colonel Hickman: I would really caution everyone not to extend these results to a general population. This information was derived from a study of USAF pilots at age forty plus or minus five years. I would be very reticent about extending the information to any other population, other country or ethnic group, and especially of any other age, because these are highly selected individuals.

Question: What is your incidence of silent ischemia in your population compared with the general population?

Colonel Hickman: The gross positivity rate of treadmill testing in our population is around 7 to 8%. If we allow for perhaps a 33% false negative rate in the higher risk index group, and a 50% positive predictive value overall (much lower, of course, in low risk index groups), we expect that we have overall about a 5% prevalence of significant coronary disease. Cardinal risk factors predict only about 40%. There are cases of coronary artery disease that we cannot account for by cardinal risk factors and until we find what the unknown factors are, we will just have to press on and do the best we can with the risk factors that we have available.

Question: Do you have any numbers about sudden heart deaths in aviators?

Colonel Hickman: Coronary events are the number one cause of death in active duty personnel apart from accidents.

Question: No, I mean the relationship between silent ischaemia and coronary deaths.

Colonel Hickman: In the Framingham study, two thirds of the people with asymptomatic coronary disease presented with an acute catastrophy. They either had an infarct or they died. We have found angiogrpahic coronary disease in a lot of aviators before they have any symptoms and we have followed them forward. They all knew they had coronary disease and knew what the symptoms of angina might be. We followed about one hundred and twenty forward, having briefed them on what to expect. In such a group of men who know they have angiographic disease, and were smart enough to listen for the symptoms, 80% of the initial symptoms were angina, and only 20% were catastrophies, and only one of those died. "Silent ischemia" in this population is actually supply-demand imbalance induced by peak exercise, and not the same as silent ischemia encountered in clinical settings. This undoubtably accounts for the better prognosis of this type of silent ischemia.

What this also means to me is that angina is being ignored, not reported, or misinterpreted in clinical patients. The middle aged man in the U.S., rather than needing a treadmill, if he is high risk, needs counselling about what the possible symptoms of angina might be. We have reported this in abstract form, and will soon have a large enough population to report the ten year result. The annual event rate in our asymptomatic aviators with silent disease is 6% per year. Eighty per cent of the events are angina.

Question: Why is smoking not included in your risk index as a risk factor?

Colonel Hickman: Our risk index was developed to predict asymptomatic coronary artery disease and not to predict actual coronary end-points. It was developed to predict silent coronary artery disease. In our data, the effects of cigarette smoking appeared to be mediated through alteration of lipids, with smoking lowering HDL cholesterol. Also, the total dosage or period of exposure is a function of age and so is covered in the age variable. If the Index had been derived from a population which had significant numbers of documented thromboses, rather than from a population with only silent anatomic disease, smoking would undoubtably have been more strongly represented.

DISCUSSION - ATHENS

General Psimenos: Now that you have managed to find a way of stratification by measuring cholesterol and high density lipoproteins, how accurate are these measurements and what factors influence these very considerable risk factors?

Colonel Hickman: The most disappointing thing about trying to do this index has been the difficulty in the accuracy and precision of the lipid determinations at our local Air Force Bases. The coeffecient of variation of the tests is miserable, and yet we are trying to use the lipids not only to give patients extremely important advice about how to change their lifestyle, we are using it to select people into flying training and we are using it to try to find out if they should have a treadmill. Yet we are doing the lipids very poorly.

One of the reasons that our lipids were being done poorly was that we didn't know we were doing them poorly. Our laboratories use a commonly available quality control service from one of our national professional organizations in pathology. They send batteries of samples to laboratories, both civilian and military, and the labs respond with test results. Unfortunately, there is no "gold standard" and results are scored against the mean results of all the labs who participate. Cholesterol and HDL are done with a variety of methods in the U.S.A., and test results are widely scattered. So, including the results of poorly performing laboratories obscures performance deviations. Also, one may not receive a report that your lipids were "out of standard" if your laboratory had only 2 or 3 tests out of 30 which were substandard, and all other determinations (electrolytes, enzymes etc) were "within standard". For example, if your lab had only 2 of 30 tests "out of standard", and 4 of 30 were needed to "fail" the survey, your lab may not be aware of poor lipid performance, especially in terms of accuracy, even though precision (reproducibility) may be quite good.

We are attempting to overcome this problem in the USAF by standardizing all our labs to the same method, and we are setting up our own quality control to be carried out by the SAM lab.

The way we actually calculate how to use the index, is that we calculate a "typical" ratio over time (you need at least 3 sets of lipids) and we calculate a typical ratio, and then use the current age. Because otherwise an individual would never be able to change his risk, if we didn't add in the new HDL. It is necessary to have a 14 hour fast.

We think that the dextran sulphate method is the method of choice for HDL, and we believe that it takes a very strong quality control program.

We have also noted that you can change your HDL very quickly. When an aviator receives notice to come to Brooks for an evaluation, he has about a six week waiting period. If he loses weight and starts an exercise program, his HDL goes up. And so, you have to know something about the individual you are examining in order to interpret the results also.

But to get back to your main question, it is a tremendous problem, the variability of these estimates. We think we can do much better at it.

Dr. Masdrakis: Which angiographic criteria did you use in your data?

Colonel Hickman: Almost all the data I showed you were 50% or greater narrowings. We consider it a significant case of coronary disease if a major vessel is narrowed to less than 50% luminal diameter because that reduces flow by two-thirds.

We call it minimal disease if there is no lesion greater than 30% and if the aggregate or sum of lesions does not exceed 50%.

Dr. Masdrakis: If you have a patient with an abnormal exercise test and a low risk ratio, and he has a normal thallium, do you perform an angiogram?

Colonel Hickman: Currently in our U.S. Air Force system, there is no way that you can fly with an abnormal treadmill test, no matter what the other circumstances are, without angiography. That is our rule, and the obvious answer to your question is that we shouldn't have done a treadmill on that person-that is the best answer to that question. But still today, we would have to do a cardiac catherization. I admit that the rational thing to do if you were not bound by a regulation, would be to say that the risk for coronary disease is so low, that we will forego the catherization. But we are not at that point.

The Risk Index is currently under an evaluation study, in the Tactical Air Command only, and until the validation is completed, we must continue to follow our current policy.

Question: Do you think that the stress of the aviator's environment is an additional risk factor for CAD? Is there a way to measure it?

Do you measure apolipoprotein, because there are some papers that indicate it is a better index than HDL cholesterol?

Colonel Hickman: Do we believe that lifestyle and stress in terms of our aviators plays an important part in the coronary disease? We have some conflicting information. Most of you are familiar with Friedman and Roseman's Type A and Type B personality. A number of years ago we had a very well trained interviewer conduct their interviews. We videotaped the interview with the aviator who was going to cardiac catherization, and we sent those off to independent experts who scored the interview with no knowledge of the cath results. The results were that we were unable to say that Type A or Type B personality had any bearing on the presence of coronary artery disease.

Drs. Friedman and Roseman are gradually refining their definition of A and B so that maybe the categories were not specific enough.

From that standpoint at least, in terms of personality type, we don't have any confirmatory information.

We do have some evidence that elevations of cortisol, and especially obliteration of the diurnal variation during stress, may be associated with increases in cholesterol, and therefore one would have to conclude that it may be a risk factor for coronary disease, although I could not say that for sure.

One of the reasons we are not sure we would want anyone else to use the Index is that we have not validated it, but also because the field of biochemical risk factors is rapidly developing. We are currently doing a blinded cooperative study with the Mayo Clinic. They are doing HDL subfractions, and apolipoproteins on aviators undergoing caths at Brooks and when we have enough subjects, we will break the code to see if they have any predictive power. We feel very strongly that with A1 particularly, there will be some predictive power.

Question: The USAF Risk Index was developed for your particular population with an age distribution of 35-45. Could you comment on and make any recommendations for risk stratification in the older population of professional civilian airline pilots?

Colonel Hickman: Our index applies only to a population of 40 plus or minus 5 years. We are certain that if our population were not that young, that age would be disproportionately weighted in our equation.

I believe that for civil aviation, it must contain some information about HDL. I think that if I had an airline captain who had an index of 6 or greater, who was

over 45, and who had any other single risk factor, I would pursue a treadmill test. However, that is a subjective feeling, because we don't have the angiographic data to make the statement. I think that someone must come up with an index for an older group of aviators, which I believe will have to contain HDL or apolipoprotein data. It will invariably have to contain age.

It may be easier to say who doesn't need a treadmill test. If you have a 55 year old captain who has a ratio of 3, a non-smoker, non-hypertensive and both parents lived into their 70's, I believe you've got your answer there - I don't think you need to do anything else.

I do think that some sort of screening is going to have to be brought to bear on the individuals who have had lifelong elevated risks and are involved as airline captains.

Dr. Masdrakis: In Greece, we started two years ago to do exercise testing as a screening test in aviators above 40 years old. We don't yet have the results, but it would be nice to use the risk ratio also in the future. What is the prevalance of CAD and the predictive value of the treadmill in USAF aviators in this age group?

Colonel Hickman: We don't know what the overall prevalance in that group is. Our estimated prevalance in those who indices over 12,000 is more than 40%, between 10,000 and 12,000 is about 25%. Those two groups contain a very small proportion of all individuals over the age of 40.

Back when we cathed all of the SVT's, bundles, VT's, which included a significant number of aviators over the age of 40, the background amount of coronary disease in that group which had an indication for cath, was 15-20%. I suspect that for those that do not have an indication, that the prevalance would be in the neighborhood of 10% of significant disease.

I hope that we will be able to have the results of your treadmill study. I think that is a critical study for aerospace medicine - a very important series of patients.

General Psimenos: We have faced a puzzling situation with our civilian pilots (we attend all of them in our Aeromedical Center). We have seen a prevalance of CAD which is about twofold that in the rest of the population. Of course, these are all above the age of 40. We have had many instances of infarcts - it looked like an epidemic. Most of them had a double career. They were ex-fighter pilots who later went to the civilian sector and had to undergo a very stressful training period and professional survival, competing with younger civilian pilots that had got their training as civilians from the beginning.

Anyway, it still is an issue, and we have decided that a stress test will be mandatory for all of them, above the age of 40. That is the group that Dr. Masdrakas is referring to; they have a different lifestyle and different stresses than the fighter pilots, and they are older. In comparison with other airline pilots we feel we have a higher incidence of CAD.

Colonel Hickman: I think if you've made a decision to go ahead and do it, I would keep on until I knew what my results were. One of the reasons that we will have a problem pursuing our Index Air-Force-wide is that in our pilots who fly for the Air National Guard, and for the Reserve, a great many of them are airline pilots. For them, a treadmill test for military duties may place their livelihood at jeopardy. They risk losing not only their military flying status, but their airline rating. Many of them may choose to drop out of the Guard because of that risk of testing. We therefore want to know that we are very precise before going Air-Force-wide.

If you are seeing a rising trend of CAD in your airline pilots, I don't think anyone could argue with the approach that you've taken, because no one has prospective data to tell you how to do it. We can tell you that in a younger population, we had a lot of people who went to cath who didn't have the disease. On the other hand, we've never had a death, nor an infarct, nor a stoke due to cath. In fact, since we have gone to the Judkins approach, in about 1977, we simply have not had a major complication of any kind, so that angiography should not be looked on as such a terrible thing. The information is extremely precise. The problem with us was that it was very labour intensive, cathing people who didn't have the disease.

Also, in North America, the last ten year trend for coronary events is going down, whereas if you have a trend that is still peaking, I think you are justified in a more vigorous approach from a diagnostic standpoint.

DISCUSSION - COPENHAGEN

Question: What is your estimate of disease prevalence in a group who risk index exceeds 12,000?

Colonel Hickman: We feel that the disease prevalence is about 33 percent in the group whose index is from 10,000 to 12,000 and it's about 20 percent in the next group below that. Overall, the prevalence is about 5 percent overall.

Question: In that high group then, the risk of false negative is not that great. You have to get up to the 80 percent level before a very high false negative rate.

Colonel Hickman: I would have to disagree. The false negative rate is a definite problem simply because there are so many candidates for the disease. Our data came from three different groups going to catherization with a negative treadmill. One was coronary calcification on fluroscopy. In such a group, that is with coronary calcification on fluroscopy, a negative thallium, and a negative MUGA, you still have about a 30 percent chance of having one 50 percent lesion on angiography. Now one reason for feeling that it should be lower than that is that you might not include people with a 50 percent lesion. If we move the definition to a 75 percent lesion, the false negative rate would be significantly lower. Coronary artery disease, though, is a progressive and capricious disease and most of our 50 percent lesions at a high level of exercise are clearly causing reversible ischemia.

Question: In Holter monitoring, do you find the ST segment of value in predicting disease?

Colonel Hickman: In an older clinical population, the ST segment in Holter monitoring is proving to be of value in the assessment of silent ischemia. However in our younger population, we have just not found the ST segment change on Holter to be of any value. What we study are asymptomatic, relatively younger men who have ischemia precipated only by high levels of exercise creating a supply-demand imbalance. Individuals with angina never make it to Brooks, but in the asymptomatic population the ST segment response on Holter has not proved to be valuable, even in the highest risk index group.

Question: The USAF risk equation is a very heavily age-weighted. With such an equation, are we not zeroing in on the population who are not active fighter pilots?

Colonel Hickman: We have 2,000 aviators in the US Air Force who are over the age of 40, and one out of every two is an active fighter pilot. We would prefer not to investigate pilots who are "flying a desk", but with the concept of state of "readiness", they are still considered on active flying status.

Question: Can you give any estimate of the incidence of coronary disease if all your non-invasive tests are negative?

Colonel Hickman: Even at Brooks, we don't cath people in whom all the non-invasive tests are negative, that is for risk factors, alone. So I can't really answer the question.

Question: Does the USAF have any epidemological evidence to indicate that their risk identification and intervention has lowered the incidence of coronary disease amongst their aviators?

Colonel Hickman: We don't have the epidemological information as yet to show that coronary risk intervention has had an impact on the disease process. We know that the gross mortality of coronary disease in the Air Force has started to fall just like it has in the civilian community. We have had a cost analysis done by Perdue University which has conclusively shown a single dollar spent on risk identification and intervention would return about $3.50 after 10 years based purely on available data from Framingham, Seattle, and Oslo.

Question: What is the value of exercise stress testing in selection for aviation screening? What is the significance of ST depression in candidates?

Colonel Hickman: I know that there are a lot of aviation centres which are doing treadmill testing on 21 year olds. We know they are very unlikely to have coronary disease and yet we are looking at them with what is basically a coronary disease test. This limits the utility of the stress test in this population.

Even for rhythm disturbances, if you take a group of young men with a high degree of vagal tone, you'll find a spectrum of rhythm disturbances most of which fall under the umbrella of a normal variant. The only way you can define the significance of rhythm disturbance is how it relates to underlying organic heart disease. If you started with a treadmill and a Holter first with a group of candidates, you would end up with a large number of non-specific findings. I can't imagine the utility of a Holter and exercise test on a healthy young man because the things I'm interested in are just not there. It's the structural abnormalities that we really want to examine, along with risk factors for coronary disease. We do not use (and I would be dead set against using) treadmills and Holters as screening tools for aircrew candidates. Why not look for structural abnormalities first, in a 21 year old, rather than doing non-specific tests like treadmills, or Holters, and then still having to launch into a search for the organic substrate?

Question: Isn't it a funny situation in the US Air Force that you can make a real mistake by doing an exercise ECG on a pilot?

Colonel Hickman: That is the entire bottom line to the Bayesian approach to screening. If you ask the wrong question of the wrong people you will always get the wrong answer. If you do the treadmill test on the wrong people, you will always get the wrong answer.

DISCUSSION - ESKISHIR

Question: You mentioned during the lecture that for the USAF Risk Index, diabetes mellitus, smoking and other factors were not found to be significant or their effects were perhaps encompassed by their effect on lipids. Do you think that stress or psychological factors related to flying might be important? Could total flying hours or other measures of flying stress be correlated with coronary disease in aviators?

Colonel Hickman: There may be an effect of stress in the mechanism of coronary artery disease, but it is extremely difficult if not impossible to measure and quantitate. In our risk equation, any effects of sustained flying stress on the development of coronary disease would to a large extent be covered by the age factor. We do know that prolonged stress may obscure the normal diurnal pattern of cortisol secretion, and that may elevate serum cholesterol.

In the 1970's, type A and B personality types were reputed to be related to the development of coronary artery disease, with a purported higher incidence in the time-conscious, aggressive, subliminally angry type A personality. USAF/SAM sent a nurse to train with Dr. Friedman and Roseman in the interview techniques of categorizing type A and B personalities, but we were unable to find any correlation between angiographic coronary disease and personality type in our aviators. Perhaps more discrete definitions of personality categorization will yield a better correlation with coronary disease.

Question: Why is smoking not a factor in your risk equation?

Colonel Hickman: We found that cigarette smoking as a non-continous all-or-nothing variable did not add significantly to the predictive of our risk equation. This is not to say that smoking is not a risk factor for coronary disease, because we know it is. But, in our equation, which applies to our unique population, the effects of smoking are encompassed in the other factors in the equation. Smoking decreases HDL cholesterol, and so increases the ratio. The cumulative effects of smoking increase with age and so are included in the age factor. The effects of smoking are buried in these two factors in the USAF Risk Index. The population of catheterized aviators from which this Index was derived contained no infarct patients. All subjects had silent anatomic coronary disease. Undoubtedly, we would have had a positive correlation with smoking if infarct subjects were in the study group.

Question: What about triglycerides as a risk factor? Why are they not included in the equation?

Colonel Hickman: Most people with elevated triglycerides have correspondingly depressed HDL cholesterols, so the effect is covered in the USAF Risk Index by the ratio. Triglycerides levels vary even more than cholesterol from minute to minute and hour to hour, making them more difficult to use in any risk equation.

Dr. Gray: One of the problems we have is the variability of cholesterol determinations done by the many different civilian labs we use across the country. There is just too much variation, especially in the HDL, for us to put a great deal of weight on the ratio, but in Canada, we still have a definite requirement to institute a uniform cardiovascular screening program in our experienced aircrew, by stratifying according to risk and doing additional screening on the high risk group. We, too, do not have nuclear cardiologic facilities readily available within our military medical system. However, I don't think any of these shortcomings should deter us from initiating a basic risk stratifying system with secondary screening beginning with a maximum exercise test. Generalized screening with exercise testing obviously is not on because of the high rate of false positives and resistance from the aircrew, but I think in smaller air forces such as ours even a simplified stratifaction procedure such as quantification of the number of classic risk factors with exercise stress testing of those with two or more positive risks would idenify many of the aircrew we now have flying with asymptomatic coronary disease.

Colonel Hickman: Reproducibility of the measurement of total and more so the HDL cholesterol is also a problem in the US Air Force. We are in the process of correcting this problem, by implementing a rigid control program through the School of Aerospace Medicine. This is especially critical for us since our Risk Index relies heavily on the value of total and HDL cholesterol, and the Index was derived on values from the School laboratory, while the screening program is carried out throughout TAC with cholesterol values from the different Air Force labs.

I would like to emphasize that the current risk factors are not those we will be using in the future, since we are unable to explain all cardiovascular events with the present cardiovascular risks. As the mechanism of coronary atherosclerosis unfolds, our risk factors will be revised to include I'm sure, apolipoproteins, and our risk equation will have to be appropriately revised, becoming ever more powerful as an indentifier.

Since in the Turkish Air Force you screen all your experienced aviators here on a regular basis, I would urge you to start collecting risk factor data now on all your pilots, for a future stratification program.

VALVULAR AND CONGENITAL HEART DISEASE IN THE AVIATOR

L/Colonel W.B. Kruyer

DISCUSSION - FURSTENFELDBRUCK

Question: What do you do with a hypertrophic non-obstructive cardiomyopathy?

L/Col Kruyer: Disqualify them whether they have obstruction at rest or not. How bad the obstruction is has really no relation to their prognosis, at least as far as symptoms, arrhythmias and sudden death go. In fact, no obstruction at rest is one of the factors in predicting sudden death.

Colonel Hickman: Until a few years ago, if we found an aviator with echocardiograph evidence of hypertrophic cardiomyopathy, we performed invasive studies to determine the gradient, and provocative studies to precipate outflow tract obstruction. About that time, a large natural history study came out which pointed out that the ability to find or precipate obstruction did not seem to affect the overall outcome in the natural history of the disease. So since then, instead of trying to pick out the ones we should ground, based on invasive and provocative studies, we disqualify all of them. The real answer is that if we were doing echocardiography as a screening tool when they were applying for training, we wouldn't have money invested in a hypertrophic cardiomyopathy who is flying.

Question: Given that the natural history of biscuspid aortic valve is generally fairly benign into the late fifth and sixth decades, what are your recommendations about aircrew applicants who are found to have biscupid aortic valve either through routine echocardiographic screening or on a case basis based on clinical suspicion followed by echocardiography?

L/Colonel Kruyer: The USAF policy now is to waiver bicuspid aortic valve in a trained aviator if they don't have significant stenosis or insufficiency. We do not accept applicants with bicuspid aortic valve into pilot training. About half of them will be stenotic to some degree by age 45, but as you mention that is getting toward the end of a military flying career. A significant number of them will also be insufficient by the third and fourth decade. Although it is not typically a malignant course in the absence of endocarditis, they may well become stenotic and we feel it is sufficient reason to disqualify them from flying training.

Colonel Hickman: If you had a small applicant pool, and you had a means of directing candidates with bicuspid valve into tanker/transport/bomber (TTB) flying, you maybe could consider accepting them. With the advent of colour-coded doppler, the probability of detecting mild AI that may not even be clinically detectable becomes higher.

In our Air Force at least, it is very difficult to switch from tactical flying to flying tankers, bombers and transports, midway trough a career. I don't think that it is justifiable to start someone off with bicuspid aortic valve into high performance flying with few career alternatives if he progresses. For that reason, we have decided not to take bicuspid aortic valves into flying training.

L/Colonel Kruyer: Another consideration is that these individuals are at risk of endocarditis, and going back to our Vietnam experience, we had quite a number of pilots incarcerated as POWs for a good number of years, and someone with valvular disease just from the extremely poor hygiene conditions, could be at significant risk of acquiring endocarditis in those circumstances. This is one of many things that you have to think about when you are considering who to let into militray flying train- ing.

Question: How quickly can hypertrophic cardiomyopathy develop? Could you miss it at age twenty and yet discover it later?

L/Colonel Kruyer: I don't know of any natural history studies that could answer that question. We have seen some cases in aviators who for some other reason had an earlier evaluation and had a normal echo, who later developed a diffuse hypertrophic process.

Colonel Hickman: At the AGARD panel meeting in Portugal in October, 1986, a decision was made to have a long-term NATO Echocardiographic Working Party. All NATO countries doing echocardiography on aircrew, either applicants or experienced aircrew, will be invited to pool their data and follow these aviators in a long-term prospective fashion to see if there are occupational cardiac health risks in high sustained G/ high performance flying. If would be reassuring if we could tell people who were taking up high performance flying that there is no unique cardiac risk involved. We will also compile other valuable data about age-related prevalences of anatomic findings on echocardiography.

DISCUSSION - ATHENS

Dr. Masdrakis: In aviators with congential heart disease, after operation, do you recommend a cath or only echo before going back to flying status.

L/Colonel Kruyer: Generally, we will repeat cardiac catherization to be absolutely sure that there is no residual defect. Colour flow doppler, when we have more experience and the world literature provides more data, might allow us to get away from that policy, but right now we are very concerned that there is no residual shunt which might be aggravated or reversed with a straining manouver with a potential for a paradoxic embolus, for example.

Question: Do you accept doppler findings for mild aortic stenosis or other congenital lesions?

L/Colonel Kruyer: We don't usually perform cardiac catherization for assessing valvular lesions now. There were catherization criteria presented for a number of valvular lesions in the lecture. Usually, we are able to satisfactorily assess the severity of the lesion and make the waiver determination non-invasively. Catherization may be necessary for an abnormal test in the routine non-invasive evaluation (treadmill or thallium) that might suggest the possibility of underlying coronary disease, in which case the valvular lesion is assessed as well as the coronary circulation.

Question: What would you recommend for a candidate with atrial septal defect?

L/Colonel Kruyer: We do not accept applicants into flying training with congenital disease, either with or without surgical repair, as a general policy. We have granted waivers for surgically corrected ASD's when the ASD was discovered in a fully trained aviator, then successfully repaired, with a confirmatory cath 6 months post-op.

Question: If you have an aviator with aortic stenosis with a gradient of 30 or 40 mm Hg, do you recommend a waiver for transport flying?

L/Colonel Kruyer: We can recommend waivers for tanker-transport-bomber (TTB) only for conditions in which the risk is uniquely related to G forces e.g. aortic insufficiency, not for mild disease that we may not want in a single-seat aircraft but may be permissible in a multi-seat aircraft. That is not our mandate from the Surgeon General. We are allowed to recommend TTB only for diseases where the risk is related strictly to high G stress. The Commanders that have the bombers and the transports consider that all their aviators must be fit to fly as well. The don't look at them as a pilot and a co-pilot who can take over if the pilot is having problems. All the crew members in the cockpit of a transport or bomber have a unique mission, and all of their roles are important to the mission, and therefore they want all of their aviators well and fit.

DISCUSSION - COPENHAGEN

Question: Do you really feel that the baboon studies showing increased aortic regurgitation with G suit inflation are applicable in the high G environment?

L/Colonel Kruyer: We can't really say for sure that these data apply. We are not ever going to be able to do the study in which we put humans in the centrifuge with a G suit and do angiography to find out whether their AI (aortic insufficiency) increases. The baboon studies are just another one of the many aspects about AI that we wonder about.

Question: Have you tested patients with AI in your centrifuge to see how they respond? The baboon studies are really misleading information.

Colonel Hickman: In years past, before we restricted aviators with AI, a centrifuge study formed part of the annual evaluation of aviators flying with aortic insufficiency. During that period when they were on active flying status, about 95 to 98 percent of them showed normal G tolerance over time. Of course there were no tools to assess the regurgitation fraction. Within a couple of years, we will have echo/doppler in the human centrifuge and we will be able to look at these people. However, they appear to have normal G tolerance. Given the natural history of the disorder, we were worried that we would intensify or hasten the natural history of the disease in the high G environment. The baboon studies were not done to study aortic insufficiency, but rather to develop a new G suit. We just felt that the data was worrisome enough that we should present it as part of this lecture series.

L/Colonel Kruyer: The concerns are not so much of the acute situation but rather the deleterious effects that might go on over a career. As I mentioned, the baboon studies were done to evaluate a new ECG-triggered G suit, but it makes you wonder if a baboon can develop 4+AI with G suit inflation, what happens to an individual who already may have some native AI under that situation.

Question: How intensively do you look for arrhythmias in valvular lesions?

L/Colonel Kruyer: Some of the valvular lesions are not associated with an increased risk of arrhythmias, while others are, for example mitral stenosis and mitral prolapse.

The standard USAF/SAM protocol includes a number of opportunities to uncover arrhythmias, including the exercise stress test, exercise thallium and MUGA study, and the Holter monitor. The Holter monitor is actually a 16 hour Holter which will uncover 80 to 85 percent of the arrhythmias that might be detected over a 24 hour period. In high performance pilots, the centrifuge study provides an additional opportunity to look for arrhythmias.

Question: What do you do with individuals who show some but not all of the features of asymmetric septal hypertrophy.

L/Colonel Kruyer: We would have to treat each case based on the individual studies, but as a general principle, we would err on the side of conservatism about letting someone fly who might have hypertrophic cardiomyopathy.

Colonel Hickman: The answer to that question is really not to take them into training. We really don't want to find an individual with a thick septum with a question of HOCM when he is 29 years old and a trained aviator. We should really find this out before we take such individuals into pilot training.

DISCUSSION - ESKISEHIR

Question: Do the dispositions you have described apply to trained aviators.

L/Colonel Kruyer: The various Study Groups at USAFSAM are for trained aviators.

Question: Why doesn't the USAF do echocardiograms on aircrew applicants?

Colonel Hickman: The main reason we do not do selection echos is that we do not have centralized screening. Our screening is spread across many facilites, and the way it is presently organized, we can not justify the costs for equipment, and for training both the extra technicians and cardiologists that we would require. Nor could we justify the manning allocation of certified cardiologists at most of these bases soley for doing screening echos.

The answer for us in the long run is that our selection screening facilities have to become more centralized. This will also allow us to learn more from our screening data.

AEROMEDICAL ASPECTS OF MITRAL VALVE PROLAPSE

Colonel J.R. Hickman

DISCUSSION - FURSTENFELDBRUCK

Question: Are aviators being returned to flying status with a waiver while taking Aspirin as an antiplatelet agent, and as a corollary to that, could you comment on the recommendation of many internists that everyone over the age of thirty-five should be taking a single aspirin a day?

Colonel Hickman: For people over the age of thirty-five there is a large matc ed cohort study going on right now at Harvard University in which several thousan US physicians have enrolled in a twenty year study. The cohort has been divided into an aspirin and placebo group. This study will hopefully answer the question as to whether vascular events are decreased by taking aspirin over the age of thirty-five. Right now I don't believe everybody ought to be on an aspirin a day but I know a lot of pathologists who do take an aspirin every day and I know a lot of cardiologists who do.

We do not have any of our prolapses actively flying while taking aspirin. The cases of asymptomatic coronary artery disease that are taking aspirin are not flying. We don't have anybody flying while taking therapeutic aspirin.

(Eds. Comment: Since the Short Course, the Preliminary Report of the ongoing Physician's Health Study indicated a significant reduction in the event rate for CAD in the group taking 325 mg ASA on alternative days, prompting an early release of this data. New Eng. J. Med. 318(4); 262-64,1988)

Question: There are two kinds of mitral valve prolapse, one being the clinically detectable type with a mid systolic click and/or systolic murmur and the other being purely echocardiographic prolapse with no clinical signs. What percent of your mitral valve prolapse study group fall into each of those categories?

Secondly, do you think they are different ends of the spectrum of the same disease, with the same complications and complication rates, or do you think that echo prolapse may have a different prognosis than clinically detectable prolapse?

Colonel Hickman: I think the relatively high early complication rate in our study group had to do with the way our patients were selected, in that we got the patients with the most severe prolapse sent to us first. We are now into a group of patients who by and large have only a mid-systolic click or echocardiograph prolapse. However, out of our study group of three hundred and some odd, we have had to disqualify one hundred and thirty-three of them. We set up criteria of what we thought mitral valve prolapse out to consist of and 355 met that threshold. We then followed them serially.

To us it doesn't matter so much whether it is a clinical syndrome or an anatomic finding. Most of those that we have had to ground for complex ectopy have not had much more than echocardiographic findings and yet they have cost us over a hundred million dollars in training costs since 1976. I think it is an interesting question for clinicins but I am not sure that I am that concerned about it. I would like not to ground anybody who has only a mild foible on his echo. I hope we will eventually learn to be able to tell these people apart, but until we can, I think it too expensive to train them.

Let me sum up by saying that we don't have enough natural history about mitral valve prolapse to know what exactly where we stand. Here is what Dr. Levine had to say about diagnosing prolapse.

"The techniques for evaluating prolapse are often combined assuming truth to be found at their intersection. The concept that truth lies at the intersection of two areas of confusion is similiar to saying that lost travellers meeting by chance can tell each other where they are."

I think that is sort of where we are with prolapse right now. On the other hand we can't just not make a decision, and in any of these natural history studies, the ground rules start off very conservatively, and then you widen them as you get more data.

Question: Although there are many patients with mitral valve prolapse who have arrhythmias, arrhythmias are also a common finding in the general population. How can we be sure they are related variables in a patient with prolapse?

Colonel Hickman: I think that life is not fair in that regard, and although there will be some normal people who will be disqualified, we found that arrhythmias were ten to twelve times more prevalent in our prolapse group. Ventricular ectopy is common, and mitral valve prolapse is common, so two common things are frequently going to be seen together, but I think from the point of view of aerospace medicine that you must conclude that it is not true-true unrelated but rather true-true related.

DISCUSSION - ATHENS

Question: What is the gold standard for mitral valve prolapse? A mid systolic click, or catherization?

Colonel Hickman: I believe it's fair to say that if they have a non-ejection click, they have prolapse. No matter what else they have, I don't know of anything other than prolapse that will give you a non-ejection click.

I think that in screening, even though we don't know exactly what mitral valve prolapse is, our goal ought to be to eliminate those candidates that everyone would hold up their hand and say "This is a case of prolapse", because even if we did that, we would eliminate about two-thirds of the really bad candidates that we have trained to fly airplanes.

The other problem is that when we say that someone has supraventricular tachycardia, and he has mitral valve prolapse, he should be disqualified. Well, SVT is common, and prolapse is common, and they both may be present and be unrelated, but I think that we have to make the aeromedical decision that they are related. When we reviewed our natural history data and saw a tenfold increase in ventricular complexity, I believe that aeromedically we have to say the two are related. So why don't we disqualify the prolapses that are the obvious prolapses and let those candidates into flying training that may have only the mild anatomic abnormality? For us, it would be far better than what we are doing now, since both subtle and flagrant prolapses are getting into flying training. But, I freely admit that we don't know what prolapse is, totally.

Question: Do you disqualify candidates with non-specific echo findings?

Dr. Gray: On the M-mode, our criteria are greater than three millimeters posterior motion. Although you can get false positive prolapse on the M-mode by angling the transducer, it's hard to produce 3mm of posterior bowing.

On the 2D, our criteria are motion behind the line of the mitral annulus on the parasternal long axis view, of either the anterior or posterior leaflet. We disqualify candidates with either or both of these findings on echo.

We do not disqualify for what we term "non-specific billowing" that is movement below the level of the annulus on the apical four chamber view.

Colonel Hickman: Dr. Masdrakas, what is the prevalence of mitral valve prolapse that you are picking up with your ears? You may have one of the only places where candidates are examined by a cardiologist, and I think it would be valuable if you could report the incidence of auscultatory prolapse in a group of candidates.

Dr. Masdrakis: We don't have that information now.

DISCUSSION - COPENHAGEN

Question: Can you provide as part of the course a booklet of guidelines as to the recommendations for standardization of screening for aircrew candidates and experienced aircrew.

Colonel Hickman: One of the aims of this course was to provide answers to questions using the current available data base, but for questions in which data were not available, we would provide an opinion. What we really need to answer that question is a long term natural history to follow individuals who are identically selected. This is one of the aims of the Aeromedical Panel's long term echocardiographic study which is scheduled to commence in 1988. We can't provide exact criteria for mitral valve prolapse at this time because there still are many divergent opinions.

Question: The combination of significant arrhythmias and mitral valve prolapse were mentioned as disqualifying for flying. Does the presence of VPB's count as a significant arrhythmia?

Colonel Hickman: We originally had a definition of significant stress arrhythmias as 10 VPB's per minute or 20 percent of the exercise heart rate, multiformity, or SVT or VT. We have now changed that to include only, VT, SVT or ventricular pairing.

Question: Could the prevalance of the various complications of mitral valve prolapse that were demonstrated at USAF/SAM have been influenced by the pre-selection of the study group?

Colonel Hickman: The mitral valve prolapse study group at USAF/SAM consisted of aviators in whom prolapse was found as an incidental finding on clinical examination on routine aircrew medicals. They did not have symptoms of prolapse and so differ somewhat from a clinical population with prolapse. The control group were aviators referred to USAF/SAM for other reasons and so had something that required investigation. If anything, this would push the control group towards the group with MVP and lessen the differences rather than magnify them. They tended to prevent over-estimation of MVP risks. For instance, the control group included over 1,000 people who had been referred for syncope who did not have prolapse.

As far as the degree of ectopy goes, I believe the difference between a true control population and our MVP group would show the differences to be even greater because a lot of the people in the control group were referred for ectopy.

The reason we don't have a better control group is that there is no way in the USAF that we can require healthy people to come for Holter, treadmill, thallium and MUGA.

Question: Are the Scandinavian Airlines doing stress testing or echocardiograms on applicants?

Dr. Alnaes: In Norway we do not do echocardiograms but we do stress testing, not so much to look for cardiovascular disease, but rather to provide an estimate of the degree of fitness and to provide a base line study for future comparison.

DISCUSSION - ESKISEHIR

Question: What are the clinical signs of mitral valve prolapse?

Colonel Hickman: The mid-systolic click is the most specific finding, and has only been described with prolapse. The click and murmur both vary in timing with left ventricular volume. Manoeuvers which decrease LV end-diastolic volume such as standing up from a supine position, or inhalation of amyl nitrite, cause the click to occur earlier in systole, and lengthen or accentuate the murmur. Increasing ventricular volume e.g. by squatting, or lying, causes the click to occur later in systole, and shortens the duration of the murmur. It is important when susculating for prolapse to examine the patient sitting, lying, standing and squatting, during slow inspiration and expiration, and following a Valsalva. Inhalation of amyl nitrite, a rapid vasodilator which transiently decreases LV end-diastolic volume, may bring out a click or murmur, and may be quite helpful if prolapse is suspected.

PULMONARY PHYSIOLOGY AND PULMONARY FUNCTION TESTING IN AEROSPACE MEDICINE

by

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DISCUSSION - FURSTENFELDBRUCK

Question: Do you recommend alpha 1 - antitrypsin levels as a screen?

Dr. Gray: In candiates with a family history of serious obstructive airways disease and emphysema we do antitrypson levels on a case by case basis. We don't do it nor recommend it as a general screening measure.

L/Colonel Rodig: We do it and we find positive tests in individuals with no pulmonary function abnormalities and no family history at an incidence of one case every one to two thousand applicants. We also discovered one case in an experienced fighter pilot.

Dr. Gray: He would be someone that you would want to follow on a more frequent basis with regular pulmonary function screening.

Question: What do you do with an applicant who has an atopic history and or bronchial provocation testing has a hyper-reactive response?

Dr. Gray: Individuals who have a positive atopic history and who are hyper-reactive on methacholine challenge testing are rejected from the pilot selection if moderately reactive to methacholine and if severly reactive, they are rejected from all aircrew selection. Our actual criteria are a minimum PC_{20} of 4 mg/dl for pilot selection, 2 mg/dl for flight engineers and navigators, and 0.5 mg for other aircrew.

Question: What about the chronic smokers who have normal lung function when they joined and they are now forty years old and flying F18s? How often should we be screening them for small airways disease? What sort of equipment should we be using in the field?

Dr. Gray: I think that smokers should be screened annually in conjunction with their annual aircrew medical for small airways disease. I would recommend maximum expiratory flow volume curves, which could be done on equipment that doesn't cost a whole lot of money. The sensitivity of the test can be further improved by adding flow volume curves before and after heliox, but for local screening, that embelishment is not a necessity.

A regular screening program would have two benefits. Firstly, it allows objective quantification of early small airways disease, and secondly it can often provide a major incentive in terms of motivation to encourage pilots to stop smoking.

Question: Do you take smokers into flying training?

Dr. Gray: Yes. Unfortunately we do. We can't reject them as yet, because of Human Rights issues in our country.

Question: Are there air forces here who reject smoking applicants?

Answer: No response.

Question: When do you you recommend body plethysmography?

Dr Gray: This will give you additional information about total airways resistance and lung volumes. Neither of these are particularly sensitive screening tests for small airways disease, but are helpful in following patients clinically and in determining the amount of gas trapping. So, we do body plethysmography only when we are dealing with patients with obstructive lung disease, identified on screening or clinically.

DISCUSSION - ATHENS

Question: How often does acceleration atelectasis occur?

Dr. Gray: I don't think it's particularly frequent occurence. We haven't done a survey in Canada, but in the US Navy, where they routinely breathe 100% oxygen on all missions, it occurs more frequently than in the Air Force where oxygen is supplied by the normal diluter-demand system. Although it is not a frequent problem, it is a situation that all fighter pilots should be briefed on and be aware of.

Question: What do you recommend that a pilot breathe when he is going to do some air combat manoevers?

Dr. Gray: My recommendation would be to use a diluter-demand regulator at a normal setting to provide whatever inspired oxygen concentration is required. I don't think there is sufficient gain from breathing 100% oxygen, which will hasten aeroatelectasis, and once you have atelectasis you have in effect a right-to-left shunt which cannot be corrected by a high inspired oxygen. I think pilots should be briefed regularly on the respiratory manoeuvers which will prevent atelectasis. The normal anti-G straining techniques if properly performed, will accomplish this.

Question: Is there a danger of oxygen toxicity when breathing 100% oxygen?

Dr. Gray: I don't think there is any danger of this in the aviation environment. The inspired oxygen tension while flying, even when breathing 100% oxygen, is still less than one atmosphere, and the duration of exposure is much too short to allow such a problem to develop.

Question: What is the primary mechanism by which small airways disease effects G tolerance.

Dr. Gray: Small airways disease with even mild airflow obstruction can create ventilation-perfusion (V/Q) mismatch with a degree of venous admixture which contributes to arterial desaturation. Increasing G levels can only magnify the pre-existing V/Q mismatch and worsen the degree of hypoxia experienced at any particular G load.

Question: How often does the provocation test need to be performed?

Dr. Gray: We do not do challenge tests as a routine screen, but based on clinical indication. We do them on any candidate with any suspicion of reactive airways disease based on history, or on our routine screening pulmonary function tests. We do them fairly frequently, so if we have a candidate with a history of recurrent bronchitis as a child, or any wheezing during childhood, or who has a history of significant upper airway atopy, we look at them with a methacholine challenge test. I haven't any exact figures, but I would saw we do them of 5 to 10% of our candidates.

Question: Do you select smokers for flying training?

Dr. Gray: At the present time, we do not reject candidates based on their smoking history. We do look especially carefully at the pulmonary function tests on smokers, and if there are any signs at all of small airways disease we reject them, but we are presently not in a position where we can reject candidates based solely on whether or not they smoke. I would like to be able to, because of their significantly increased risk of developing not only small airways disease but also coronary artery disease. I think that as a group, AGARD/AMP should make a very strong recommendation to our operators that we reject candidates who smoke.

DISCUSSION - COPENHAGEN

Question: What is the sensitivity and specificity of the various tests that you use?

Dr. Gray: The sensitivity and specificity of flow-volume curves on air and the single breath nitrogen washout test have been looked at in a number of studies in which the results of these tests were compared with histopathologic findings in surgical specimens. The sensitivity and specificity are in order of 70 to 80 percent, comparing functional abnormalities with histopathologic evidence of small airways disease. The most sensitive tests are the expiratory flow-volume curves on air and heliox, comparing the flow rates at 50% vital capacity, and the single-breath nitrogen washout looking at the closing volume or derivative such as the closing capacity, and the slope of phase III.

Question: What are the confidence intervals that you use for defining abnormal?

Dr. Gray: As is generally accepted in pulmonary functioning testing, values beyond plus or minus 20 percent we consider abnormal.

Question: What is the reproducibility of the test such as the single breath nitrogen washout test. We find the reproducibility of such a test to be rather poor, in order of 15 percent?

Dr. Gray: I think that if particular attention is paid to detail about how the test is performed, including careful monitoring of expiratory flow rates, the single-breath nitrogen washout test is much more reproducible than that. I have

studied subjects repeatedly in a number of altitude studies, and I found the curves to be highly reproducible if performed properly.

Question: Since the sensitivity and the specificity of the tests are in order of 70 to 80 percent, we learned yesterday that they should be applied in a population where the disease prevalance is at least 30 to 40 percent. Do you apply such tests to all aircrew candidates?

Dr. Gray: We screen all aircrew candidates with flow-volume curves on air and heliox. In candidates with a history of cigarette smoking, or bronchopulmonary problems during childhood, if we find significant abnormalities on the flow volume curve on air and heliox, we then go on to do further tests including a single breath wash nitrogen washout test, diffusing capacity, and if indicated, an airway challenge test. In other words, we stratify our pulmonary function assessments based on the probability of disease. I would estimate that in the group undergoing second order screening, the prevalance of mild disease is probably 40-50%.

Question: Do you accept candidates who smoke?

Dr. Gray: I think that there is a general agreement among NATO countries that smokers ought not to have a million dollars invested in them for aviation training. I think that there is a general strong feeling that that's true, but yet it's virtually impossible in most countries to accomplish that end at present. I believe it will happen.

Dr. Vinaes: In Norway, we tend to exclude smokers but if we have the occasional candidate who passes all our ventilation tests with flying colours we take them.

Dr. Holmgren: The point I'm trying to make is just to thank you for teaching us that we should stratify material when we start to analyze it. The obvious thing would be to give them (the candidates) oxygen in the centrifuge, and study a group of smokers and study a group whom you identify, or say you have, with small airways disease whatever that is, and another healthy group. This must have been done.

Dr. Gray: This is a study that hasn't been done and is just waiting to be done. That is, to see if a group of subjects with documented small airways disease have a difference in oxygen saturation under a given level of G, and a difference in G tolerance compared with a group of normals. Both these are relatively easy end-points to measure.

Dr. Holmgren: This means that all of what you have told us this morning is pure philosophy.

Dr. Gray: Changes in oxygen saturation and oxygen tension reflecting increased ventilation-perfusion imbalance with exposure to G are not philosophy, and have been well documented. The fact that small airways disease creates ventilation-perfusion mismatching and causes hypoxia is also a well known fact. The question that remains to be answered is whether subjects with small airways disease have a reduced G tolerance.

Dr. Holmgren: You see the danger about telling us about the Bayesian approach.

Colonel Hickman: I think that Bayesian problem is much more serious if as a result of the test you have to engage in more testing which is dangerous, expensive, or involves radiation exposure. If you have 300 applicants for flying training for each position, you are in a buyer's market. I don't think it matters much if you exclude some people on the basis of testing which may not be totally specific. That may not be totally democratic but if I am in a buyer's market, I can use the test and take the Bayesian aspects of it a little more in stride.

Question: The repeated high G exposures that have such a significant effect on lung physiology, over a life time of exposure do they also have a permanent effect on lung anatomy?

Dr. Gray: As in the heart, we do not yet know what the effect of long term repeat exposure to high sustained G in the lung are. There certainly aren't any studies published as of yet, and we don't even know as yet the effects on the heart.

Question: Haven't the astronauts been studied? They have coped with high G forces.

Dr. Gray: They take it all at plus Gx, and they only take about $+3G_x$ for transient periods of time. They are exposed to only modest G forces, not similar to the recurrent high levels experienced by career fighter pilots.

Colonel Hickman: The only inferential data on which there are any long term effects from high sustained G is from the University of Utah. They did a study in our centrifuge showing persistent T loop vectorcardiogram changes. Dr. Forlini showed that, for up to 6 months after high performance profiles, there were non-specific changes. These were not corrolated with any pathologic specimens.

Minature swine, of course show subendocardial hemorrhages believed to be unique to that particular species and due to an autonomic imbalance.

The French study which showed right ventricular enlargement in Mirage pilots was not controlled for exercise. A lot of people who work in "G" say that if there

were long term deleterious effects, why haven't already seen them? Well I don't think that anybody has made a definite systematic analysis of autopsy victims for that kind of evidence.

Question: In your larger population of pilots, there must be a lot of heavy-smoking pilots. Have you encountered any problems in this group that you haven't seen before?

Colonel Hickman: The difficulty with doing that study is that the only people that are referred to us at Brooks are people with medical problems who are grounded. There are ethical barriers to placing them in the centrifuge for reasons other than why they were referred. To take Air Forces pilots in an unselected manner and to subject them to research a protocol simply because they are smokers would not pass a human ethics committee that I know of in the U.S.

Dr. Holmgren: So then you don't know if smoking has any real impact.

Colonel Hickman: Given the rules we have in the U.S. it is very difficult to do some of this work. I think that the ball should be picked up by some of the other NATO nations where they are able to do these things.

Question: How high a "G" would you need to test to show the effect?

Colonel Hickman: If you were going to do the study, you would want to have some G profiles that would give you operational information. The peak G for the F16 is 8 1/2 to 9 G and I think that you would want to know 9G's for 15 seconds as a minimum piece of information.

Dr. Sandoe: But you could calculate statistics on how many heavy smokers you have in the Air Force flying high performance, and they still pull G with no problem.

Colonel Hickman: The pulmonary system is not the only sub-system involved when you are looking at a situation where tachycardia, increasing hypotension and stagnant hypoxia are superimposed on small airways disease. That's why if you theoretically know what size coronary lesion becomes critical under those circumstances it becomes rather frightening.

I think that it is very easy to sit back and say "We should do this and we should do that and how well do you know this and how well do you not know that." But, I don't think that we need to make any apologies about areas in which we are ignorant. I think that we should apologize 10 years from now if we are still ignorant in some of these areas, but I think that anybody who has got the courage to do some testing in healthy people and try to make some long term sense out of the results has to be given credit for picking up the ball and making sure that we don't remain ignorant in those areas. I think that all of us who have the capabilities of doing these studies should undertake them and pool the data and get the information about specificity and reproducibility.

Colonel Jessen: We all know that among the population of high performance pilots there are those that never pull high G because they are not able to do it. That's known within the family of pilots, and one reason might be because they are smokers.

Another comment is that within the fighter pilot group there are pilots that come to us saying "What is going on with us, we don't feel well". I know pilots leaving the Danish Air Force and going to SAS who tell me that they are in the process of developing something they don't like and which we must follow-up.

Dr. Gray: Our countries spend millions of dollars developing life support equipment including rapid onset high G valves and new types of anti-G suits that might give a pilot an extra one-half or one G in terms of G tolerance which, in a tactical situation they (the pilots) feel may make the difference. If on the medical side, we can find a means of giving a pilot an extra half or one G, by selecting people who have lungs that will give them optimal ventilation perfusion matching, then I think we should do so. I think that the comment that this hasn't yet proved except on a theoretical basis is valid, but I think the theory has some validity. The studies need to be done.

Dr. Alnaes: I fully agree with what Colonel Jessen says about some pilots avoiding pulling G but on the other hand you should give pilots credit for behaving in ways which do increase their G tolerance. For example, there are possibly in our country no other group of professionals with a lower incidence and a lower prevalnce of smoking than high performance F16 pilots. In fact in our country there are so few smokers among them that the incidence is lower than in young doctors, which is the lowest, apart from missionaires, that there is in the count-y. Of course, that may come about because pilots do notice that if they smoke and pull G, they don't feel well, and so they quite smoking.

Colonel Hickman: In the US Air Force Academy 20 years ago about 20 percent of the cadets smoked. Today in the US Air Force Academy, less than 3 percent smoke.

Dr. Holmgren: If you use the inert gas technique to study V/Q distribution, and you do it in the post-operative period, even in heavy smokers, you see very little shunting. And if you look at people with asthma you see very little shunting. If

they shunt, we don't believe it is active airways disease so could it be that the peripheral airways are stabilized in these patients, and they have less tendency towards atelectasis than others?

Dr. Gray: I would have to disagree with you. In the post operative period in patients with small airways disease and reactive airways disease there is definitely an increased alveolar-to arterial oxygen gradient. While there may or may not be an increase in shunt, the fact that the (A-a) gradient increases is a reflection of increased ventilation-perfusion mismatching.

Dr. Holmgren: Yes but that is a different thing. I'm talking about shunting now that is a fixed shunting. We have done hundreds of them.

Dr. Gray: I think that extrapolating that kind of information from the operating or recovery room in sick patients to the high G environment is a difficult one.

Dr. Holmgren: Yes but you don't know what is happening in the small airway, whether it is more susceptible or less susceptible to G loading. If you have a small resistance in front of the small airway, it might stabilize it.

Dr. Gray: Theoretically that is conceivable, based on resistance loading studies. However, you can't escape the fact that small airways disease, whether it's fixed or reversible, does create ventilation-perfusion imbalance even at rest, and it is difficult to imagine that this doesn't become worse in a high G situation.

Dr. Christensen: We have examined lots of patients in our low pressure chamber and we have found that if patients have an FEV in one second lower than one litre they will have problems in a commercial airplane where the pressure is about 8000 feet and they will have an oxygen tension of 4 to 5 kilopascals or 30 to 35 millimeters of mercury. So we have said that such patients with a FEV1 of less than 1 litre per second should not use a commercial airplane. Patients with reduced lung function but normal blood gases will also fall more markedly in saturation but that depends where they are on the oxygen dissociation curve. If you are a patient with a lowered oxygen tension that is more than 9 kilopascals, you could fall to 4 or 5 kilopascals. If you have a patient with normal blood gases, it is no problem to be exposed to an altitude of 8000 feet. If you have lowered blood gases and an oxygen tension of less than 9, you may have a problem. You may expose those patients to syncope.

Dr. Gray: This is a valuable application of an altitude chamber. If you don't have one available, you can get the same kind of information with a low oxygen gas mixture, and from either source this is useful information to an airline medical director in deciding whether or not a patient requires supplemental oxygen during flight.

AEROMEDICAL DISPOSITION OF PULMONARY SARCOIDOSIS CHRONIC OBSTRUCTIVE LUNG DISEASE, REACTIVE AIRWAY DISEASE AND SPONTANEOUS PNEUMOTHORAX

by

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DISCUSSION - FURSTENFELDBRUCK

Question: Does the RAF approve the use of terfenadine or astemizole in aircrew?

G/C Hull: We do approve terfenadine in aircrew, but we have rejected astemizole because of its accumulation. It is a very long acting drug and takes probably several weeks to build up to its therapeutic level, and again a matter of weeks for it to decline should you wish to discontinue it. What is the position of the German Air Force? Is terfenadine allowed?

L/Colonel Rodig: No, it is forbidden.

Question: (USAF Flight Surgeon). We frequently see people who develop reactive airways disease in the Rhine Valley who give no history of such problems before. I have not had the opportunity of following such patients after they return back to the United States, and I wonder if others here have had the same experience, and what the outcome of such a problem is on return home?

G/C Hull: Typically, these patients do have abnormal immune systems, in other words, they are atopic individuals with demonstrable abnormalities in IgE and so on, and usually they develop other allergies given time. So, although they claim that it is specific to one particular area, nevertheless, they are more inclined to develop other allergies, and I don't accept that it is atmospheric pollution of an industrial nature. More often, I think it is due to some organic component in the surrounding ecosystem.

Question: What do you do with recruits who have a positive family history, and with allergic pollinosis but who have had a very successful desensitization?

G/C Hull: Hyposensitization is in decline in the UK and has been dealt a further blow by our drug commission which has deemed that it can be carried out only in locations which have facilities for full cardio-pulmonary resuscitation. They have thus indicated that they think it is of very little value and of considerable danger. I think that hyposensitization would not be accepted in any way as being in the candidates favour in terms of selection.

Question: What is the approach of the RAF to aircrew other than pilot who have historically mild reactive airways disease, and on objective testing demonstrate only mild or moderate airways reactivity, and use an inhaled bronchodilator on an as required basis?

G/C Hull: I think I would be worried about anyone flying in high-performance aircraft with any significant degree of airway sensitivity. To continue that contingency to other aircrew flying in transport aircraft would be certainly unrealistic in an air force such as ours. We should simply be overruled if we tried to be too strict with the other aircrew. Therefore, the kind of person you described flying in a transport aircraft I would think could continue flying under supervision. I am aware that this approach could be criticized.

DISCUSSION - ATHENS

Question: What is the prevalance of sarcoidosis in aviators? Is it higher than the general population?

Dr. Hull: I believe not. Aircrew are subject to more intensive investigation than the general population and many of the cases of which I told you were in fact detected on routine chest x-ray. They would otherwise never have been detected since they never complained of any symptoms.

Question: Dr. Gistromanolakis, did you have any cases in Greek aviators in the past 10 years?

Dr. Gistromanolakis: As far as I can recall, we didn't have any cases in pilots. There is a difference between countries, and although we don't know the cause, it is possible it is related to inhalants (pine pollens, that sort of thing). There were seven cases reported by Dr. Almaes from Norway which is a large number in relation to

the size of the Norwegian Air Force.
Why is there such a delay in returning them to flying duties?

Dr. Hull: The reason for the delay is the great fear of cardiac sarcoidosis and the possibility that this might be quite undetectable on initial examination and yet might develop in the subsequent year or two. And so, this rather arbitrary period of at least a year after remission has been selected as a compromise between the extreme position which has been stated by at least some chest physicians in our country that anybody who has sarcoidosis should never fly again, and the more practical consideration that important career considerations are at hand and we simply couldn't afford to waste all these pilots if they do not have cardiac sarcoidosis. So, I think the delay is arbitrary and is due to the fear of cardiac sarcoidosis.

Colonel Hickman: In the USAF, we had all our cases of sarcoid referred to Brooks so we could follow them in a serial fashion. We have about 80 cases enrolled now. The study group was started because of our concern about myocardial sarcoid. At least, given the present technology, which revolves around two-dimensional echocardiography, Holter monitoring for rhythm disturbances, and thallium scintigraphy for fixed perfusion defects, it has been a rather unrewarding search. Either there is not very much of it in the kinds of people who have sarcoid discovered in an incidental fashion on a chest x-ray, or else we are not finding it. We are now into the ninth year of this study and they are doing inordinately well. Even the small group which must be less than 3% which we have grounded because of perfusion defects, nothing has happened to them either, so our worries about myocardial sarcoid have not been borne out and our initial waiting period of two years has now been reduced to the period of time that it requires for radiographic clearing to take place. The gallium scans were not helpful for us. We are sure that we have one case in which there is a matched gallium and thallium defect in the heart, but we have stopped evaluating our aviators with gallium because some residual uptake in the hilar areas is routine, and it is a high enery isotope. We are much more reassured about the wisdom of returning people with pulmonary sarcoidosis to flying status than we were when we started doing the study. However, we are also not sure about what size of granulomata can be discovered by our current tools. But, our rules are much less strict than they were.

Dr. Gray: We, as well, haven't found any evidence of myocardial sarcoid in the pilots that we're following in Canada who have presented with asymptomatic radiologic sarcoid. We follow pretty well the same work-up as at USAF/SAM with a thallium study, Holter, and exercise stress test looking for arrhythmias. Our policy has been to ground pilots only for six months, and to do the non-invasive cardiac work-up towards the end of this period. There hasn't been any evidence of myocardial sarcoid in the six or so that we are following, and we have returned them to fly "with or as copilot" for another six months. At the end of a year, we repeat the non-invasive work-up and then let them go back to unrestricted flying.

G/C Hull: Well apart from the one case I told you of, neither have we, and we are very happy with that. In fact, the period of grounding has been somewhat shortened, and the main delay is waiting for the pulmonary disease to clear, because we believe we may encounter artifacts in the isotope studies particularly, if there is appreciable active disease still present within the thorax. Once the cardiac investigations are completed and are satisfactory, then return to restricted flying duties is usual. A further year after that, there is a reassessment prior to return to full flying duties.

Dr. Gray: I mentioned yesterday the case of a commercial airline pilot in Canada who recently presented with a new right bundle branch block, and who turned out to have pulmonary sarcoid, so it was presumed that he had myocardial involvement.

Question: How much can you rely in the the follow-up of sarcoidosis on the carbon monoxide diffusing capacity (DLCO) in case you don't have any chest x-ray findings and you see that there is a little impairement of the DLCO?

Dr. Gray: I really don't think the diffusing capacity is a very sensitive way of following interstital disease. There has to be fairly advanced interstital disease before the diffusing capacity becomes impaired, and there is a fair amount of variability in the measurement of the single-breath diffusing capacity. I think a more sensitive way of following interstital sarcoid on a regular basis is the angiotensin converting enzyme which is usually elevated if there is active interstital sarcoid.

Colonel Hickman: We have done the diffusing capacity every year that our patients have been back. Like most of the studies in these mild, incidentally discovered sarcoids, they have been unrevealing.

G/C Hull: I'm sure that would be true. Pulmonary parenchymal disease would be quite exceptional for an aviator. Nearly all of ours had very trivial if any lung shadowing which resolved extremely fast. Usually, it's just a problem of hilar lymphadenopathy.
Colonel Hickman: This does bring up the issue about how often we are doing chest x-rays in our different countries for flying physical exams. We are still doing them every two years now. How often do you believe that we in NATO really need to do

chest films? We obviously wouldn't have discovered any of these cases of sarcoid had we not been doing them, nor some cases of metastatic carcinoma of the testicle, or hamartomas. But on the other hand, we are doing an enormous number of chest x-rays.

G/C Hull: Our position at the moment is the same as yours, but there are strong moves to abolish routine radiography altogether. In fact the pendulum may have swung too far the other way, so there is even a move to abolish the initial chest x-ray. I think that would be a mistake, but I do believe that we shall very shortly find that routine chest x-rays are only required at entry and possibly at some landmark such as 35 and then in later life perhaps from 45 onwards with greater frequency.

And of course one must remember that occasionally cardiovascular abnormalities are discovered simply a result of a routine chest x-ray, so I hope we shan't abandon it altogether. But I am aware of the criticism that we are irradiating a very large number of people for a very small yield.

Dr. Gray: In Canada, we do them initially and then every other year up to the age of 40, and annually after that, so we are probably still doing it more than we need be perhaps. But every once in a while something turns up that reaffirms my belief that we should do it every other year. For instance, this year, the chap who was selected to be leader of our demonstration team was found to have Stage II sarcoid on his routine chest x-ray. So, I think that every two years is still a reasonable periodicity.

Dr. Giatromanalskis: I have looked at the chest x-rays at the Hellenic Aerospace Medicine a few years ago and I found 0.3% abnormal, mostly due to infectious diseases including tuberculosis. For that reason, we carry on doing the miniature chest-xray, which of course has a little disadvantage because sometimes we miss little lesions especially in the mediastinum, and the radiation is a bit higher. But we consider this necessary to be done for pilots every year, and for the rest of the ground people every two years, and for the recruits when they join the army or air force, and when they leave. We do find tuberculous lesions, but as far as malignant disease is concerned, I don't think there is much benefit from a chest-xray.

Colonel Jessen: The Danish Air Force had until a year ago routinely done x-rays every third year. Just a year ago we decided not to do it any more. We only take one at entrance, and after that only on indication. We are aware of the many case reports that we might have, but overall, we decided that the cost-benefit was so low that we had better be a little more specific on the indication.

Question: What about the use of alpha-1 antitrypsin in selection, or in the assessment of experienced aircrew?

G/C Hull: It does seem to me that this is such a rare abnormality that it is hardly worth screening for in individuals who are clinically normal.

Dr. Gray: I wouldn't recommend it as a routine screen either. If there is a strong family history of emphysema, or if there is evidence of significant airways obstruction and small airways disease on screening pulmonary function tests, I think that it is worth screening that individual candidate.

In experienced pilots, again I think it is worth doing only on indication.

Dr. Giatromanolakis: As far as chronic obstructive lung disease is concerned, would you recommend the use of bronchodilators?

G/C Hull: If the disability is sufficient to require bronchodilators, and if the individual concerned is one of the cases that responds significantly to bronchodilators, I would have serious doubts about returning him to flying duties.

Question: What about the use of the new antihistamines in aircrew?

G/C Hull: So far we have very little experience. I hope that it's not going to be unhappy. The manufacturers themselves do say drowsiness may be a problem and they have a number of actual reports of this. Their advice is that extreme caution should be used in operators of dangerous machinery which I am quite sure includes airplanes. As with all these drugs, I'm sure the most important test is the individual test. This is to say, it must be the case not only that the drug controls the disease to an acceptable level, but that in the particular individual, there are no subjective or objective ill-effects. I think that the onus is on the flight surgeon concerned to demonstrate this to his own satisfaction before he returns an aviator to flying duties whilst taking the drug.

Question: Regarding pneumothorax, do you expose pilots in a low-pressure chamber before you allow them to fly after surgery (pleurectomy)?

G/C Hull: No, we do not. It is thought the results of the surgery are so satisfactory that that isn't required. A chamber ride has often been suggested as a test of cure. Our attitude is that the results of such a test would be rather limited. Clearly, if a pneumothorax was induced, this individual would not be acceptable, but I think that that would be the first and the last test that Farnborough allowed us to do in their chamber. The value of negative result, however, would be very small and wouldn't allow us to change our decision.

Dr. Gray: In Canada, we do run our aircrew up in a chamber after a pleurectomy for pneumothorax. We do an explosive decompression and a rapid decompression. The value of this procedure may be strictly to make us feel better about letting them go off flying again.

Colonel Hickman: In the US Air Force, our practice regarding pneumothorax runs counter to every shred of available information including our own. We have, as the Royal Air Force has, been struck with the recurrence rate, and the impressive opposite side occurrence.

Unfortunately, we do not require any surgery, or sclerosing agents, but only resolution clinically of the pneumothorax and a chamber ride with an x-ray made at altitude in the chamber.

I am even concerned about pleurectomies that are only unilateral, given the rather impressive opposite-side recurrence rate. Your only other choice is to ground them for a period of time that essentially means the end of a career.

DISCUSSION - COPENHAGEN

Question: What are you recommendations regarding the frequency of routine chest x-rays in aircrew?

G/C Hull: Currently, the Royal Air Force does a chest x-ray on initial entry into flying training, and then every five years thereafter. The USAF frequency is every two years. What are your regulations here?

Dr. Alnaes: In Norway, wo do one on entry into flying training, on return from flying training in the U.S., and again at age 40. We do not have a specific policy, however.

Question: Is there a relationship between pneumothorax and high performance flying?

G/C Hull: Not that I am aware of. There are anecdotal reports as you can imagine but I am not aware that these are related to high performance flying per se. There have been incidental reports of pneumothorax occurring in altitude chambers so it does appear that a gradual decompression as in flying may be associated with the development of pneumothorax. How great the danger is we don't know. It appears to be relatively small, but we don't know about explosive decompression, which would presumably be a much greater stress, and to which military aviators are particularly prone.

Question: You mentioned the use of antihistamines in aircrew. Could you comment on any recommendations you have for the use of these.

G/C Hull: There has been a great deal of experimental work done at Farnborough on the various new antihistimines which are claimed not to have sedative effects. Topical treatments are accepted as safe for any of our aircrew who suffer from hayfever, and terfenadine as a systemic antihistamine has been deemed by our people at Farnborough to be reasonably safe and has been accepted as a matter of policy for use in hayfever.

Question: What about Hismanol (astemizole)?

G/C Hull: That was felt to be less suitable because it is accumlative, and very slow acting. In fact, the accumulation is quite alarming for a period once you have started treatment. Similarily the decay in blood level is very slow. Therefore the Royal Air Force has approved the use of terfenadine, but not astemizole.

Question: If you have some evidence of emphysema or obstructive airways disease have you some standard where you ground pilots, judged from spirometry, or oxygen tension?

G/C Hull: We don't have a fixed standard. We do search for abnormalities in blood gases, and if detected, I doubt if that individual would be allowed to fly in an unrestricted single seat capacity.

Question: Is ipratoprium bromide an acceptable inhaled treatment for asthma in aircrew?

G/C Hull: We don't have a specific regulation, but I don't think that it would be generally acceptable. In general, inhalation treatment is limited to cromoglycate preferably, and possibly inhaled steroids, although I think there is some concern about the absorption of the newer inhaled steroids, which are much more concentrated.

Question: Many pilots report that their symptoms of hayfever and allergies resolve completely as soon as they get into the cockpit. Can you confirm this?

G/C Hull: No I am afraid I can't.

Colonel Hickman: One of the cases presented by the Hellenic Air Force was of an Olypmic Airlines pilot who had an attack of asthma during flight over the Mediterranean. In most cases there may be no antecedent evidence that pilots have problems

during flight with atopic symptoms. Based upon that kind of history, virtually 99.9 percent of all aviators who are grounded with high grade severe obstructive coronary disease have never had any problems in flight. In fact, they didn't even know they had severe obstructive coronary disease until we investigated them. In aerospace medicine, we must always assume that the disease will be present during aircrew duty performance.

When I hear the reference to the fact that they "never had a problem while flying" the thing that comes to mind is that the history of aircraft accident investigations reveals that most aircraft accidents now are due to a confluence of very rare improbable events that coalesce at one point in time and lead to disaster. If we are not ready in aerospace medicine to deal with very small probabilities, we are beyond the ability to make any impact, because most of the major issues in aerospace safety in terms of airplanes and in terms of medical qualifications have already been settled. However, we still have an alarming number of aircraft accidents that are all based on rare and improbable events.

That is basically all that we have been dealing with in my Air Force career. I have stood at the edge of enough smoking holes and helped to pick up the little bits of flesh out of those holes so that I am very sensitive about rare and improbable events. That's why the history that "nothing ever happened during flight" may be a piece of information, but it doesn't change the basic premises by which I operate in aerospace safety.

Question: If a pilot has undergone a succesful desentization, would you still have him restricted?

G/C Hull: Yes, I would have serious reservations about that. First I'm not sure how one would define a successful desentization in a man whom for 49 weeks of the years has no symptoms at all. You would have to wait obviously for several seasons and I think that even those who are enthusiastic about desentization agree that further desentization may be required later. Desentization is going through a bad period in our country of late, because the Committee on Safety in Medicine has deemed that it is such a dangerous form of treatment, with something between 17 and 25 deaths annually attributable to desentization, that they now say that the procedure can be carried out only in an environment where there are full resuscitation facilities available, that is, a hospital with full intensive care facilities. This has made desentization very unpopular. I think also that although some hayfever sufferers do report improvement, it is a much more dubious form of treatment for bronchospasm.

Colonel Hickman: Regarding sarcoid, because of the RAF Vulcan accident at Chicago where the pilot was found to have had asymptomatic myocardial sarcoid which was felt to have possibly played a role in the accident, along with accidents involving U.S. Army helicopters and 2 USAF accidents, the USAF became very concerned about the problem of sarcoid, and developed a detailed investigation policy. We followed 70 cases, initially with a mandatory two year grounding policy. In 70 cases, there was only one who had a fixed perfusion defect on thallium that was thought to be possibly due to sarcoid. We now feel that the prevalance of myocardial sarcoid is very low in Stage I sarcoid, and now require grounding only until radiologic clearing has occurred.

DISCUSSION - ESKISEHIR

Question: What frequency do you recommend in the follow-up of sarcoid?

G/C Hull: We would recommend an annual follow-up period, which might be stretched to biannually after a number of years.

Question: What is the risk of recurrence in sarcoid?

G/C Hull: I believe the likelihood of recurrence is very small indeed, but should it recur, it would almost certainly be within the first two years.

Question: What is the mechanism of antihistimine therapy in reversible airways disease?

G/C Hull: It is thought that histamine release by mast cells may contribute to the mechanism of bronchospasm through an immediate hypersensitivity (IgE) reaction, and that antihistamines may ameliorate this effect.

Question: What is you disposition for experienced pilots with reversible airways disease who smoke?

G/C Hull: In the presence of reversible airways disease, this would give us clear grounds for removing a pilot from flying duties until he had stopped smoking, and hopefully demonstrated an improvement in his small airway function.

Question: Does high sustained G cause pneumothorax?

G/C Hull: Pneumothorax in the air is a rare event indeed, although pneumothoraces have been known to occur in altitude chambers. To my knowledge, the incidence in fast jet pilots is unknown, although intuitively, one might expect an increased incidence based on the pulmonary stresses.

Question: Do you recommend screening for alpha-1 antitrypsin deficiency?

Dr. Gray: No, only in candidates or aircrew in whom there is a strong family history of chronic obstructive pulmonary disease, or in whom there are clinical features suggest such a diagnosis.

G/C Hull: In Turkey, how frequently are chest x-rays done on aircrew?

Colonel Dengiz: We do minifilms annually, because pulmonary tuberculosis is still a relatively common disease in Turkey.

Dr. Gray: What is your approach to applicants and aircrew with wheezing?

Colonel Dengiz: We reject candidates with any history of asthma in childhood. With experienced aircrew, we would ground them if at all severe.

HYPERTENSION IN THE AVIATOR

L/COLONEL W.B. KRUYER

DISCUSSION - FURSTENFELDBRUCK

L/Col Kruyer: Since our country appears to be one of the most restrictive in our approach to hypertension, I would like to ask you as representatives of other NATO countries, what your approach is to hypertension, including what drugs you use, what restrictions you impose, etc. Our regulations say that a blood pressure greater than 140/90 is hypertension and aeromedically it is disqualifying. The blood pressure has to be below 140/90 or aircrew are not qualified to fly. Treatment can be with non-pharmacologic methods, or with thiazides. If blood pressure can not be controlled with either of these ways, aircrew are disqualified from flying. What blood pressure is disqualifying in your various countries?

L/Col Rodig: Our policy so far is that if we get someone with mild hypertension or borderline hypertension, e.g. 160/90, then he gets non-pharmacologic treatment from his own Flight Surgeon and is still allowed to fly. He is given four to six months to normalize his blood pressure with a non-pharmacologic approach. If this does not reduce his hypertension, then he has to go to a medical treatment. We use thiazide diuretics and in some cases (for helicopter and transport pilots but not for jet pilots), we use beta blockers, primarily atenolol, because it does not cross the blood brain barrier and it is more or less cardioselective.

L/Col Kruyer: Beta-blockers as a class, and Inderal in particular have an adverse affect on lipid metabolism. However, as you point out, atenol has less of an effect.
So in Germany the cut off point is 160/90, and if you can control them below 160/90 with no pharmacologic means they are allowed to fly?

L/Col Rodig: Yes. I think you are right with your point of view that captopril, and the angiotension converting enzyme inhibitions will have a very good future in aviation medicine.

L/Col Kruyer: Have you used them yet?

L/Col Rodig: We have only in one case so far so I can't comment too much.

Colonel Hickman: When you place an aviator on beta-blockers, do you do any studies to try to reassure yourself that a case of silent coronary artery disease is not being obscured by being treated with beta-blockers? Do you do a treadmill test or other studies?

L/Col Rodig: Before we begin pharmacological treatment, we investigate for renal hypertension or other forms of secondary hypertension. If he has essential hypertension, then we make sure that he does not have coronary artery disease. Afterwards, he gets (centrifuge) training and then a treadmill test with medication before he is allowed to fly.

L/Col Kruyer: Even if you are just putting him on a diuretic, do you still do the coronary evaluation?

L/Col Rodig: Yes.

L/Col Kruyer: Do you ground them for a short time after you start the evaluation?

L/Col Rodig: Yes. When a pilot starts to take medication, the flight surgeon gives us his impression about the effects of the medication and about side effects, and afterwards he comes to the Institute where we do an evaluation and, if he has improved, then he is waived to go back flying.

Colonel Hickman: Once you start to treat, is 160/90 still your goal?

L/Col Rodig: Our goal is towards 140/90 but our cutoff point is still 160/90. During exercise in a submaximal test, our cutoff point is 240/120.

L/Col Marshall: The Canadian story is exactly the same as yours (USAF). If we discover hypertension (over 140/90) we ground them and get them under control, using thiazide first and then they can go back in the air. Other modalities are used with the hope of getting them off thiazides.

Dr. Gray: In Canada, we also use beta-blockers, and we recommend atenolol although propanolol is permitted as well. If beta-blockers are required, pilots are restricted from tactical fighter operations and from helicopter nap-of-the-earth operations.

L/Col Willms: It also depends who they see first. As an Internist I would recommend a non-pharmacologic approach first. I would get them to see the dietician, to get into an exercise programme, and I would explain what the risks are, because my clinical experience with hypertension is that people have been told that they are hypertensive or borderline hypertensive but no one has ever sat down and talked to them long enough to make them understand why we are concerned about this. Very few patients know that twenty years down the road we may be treating them again, but this time for the complications. There are much more amenable to treatment with non-pharmacologic means if you tell them what is going on.

Colonel Hickman: I think one of the mistakes we have made in the USAF, (and I think the policy will be changing soon), is that once we have established a diagnosis of mild hypertension based on preponderant blood pressures, we have grounded the aviator while we began treatment. The effect of that was to cause the local flight surgeon to go to a pharmacological mode of therapy much more quickly than I think was good for the air force or the patient. Grounding them to initiate therapy tended to de-emphasize a somewhat slower non-pharmacological approach. There is very little aeromedical risk in allowing an aviator to fly who has a blood pressure of 170/95 for a short period of time.

L/Col Marshall: I would agree with that but you could also argue that you certainly get the aviators attention by grounding him initially and you are going to get better compliance down the road in controlling his weight, fitness etc.

Colonel Hickman: Well that is the other side of the coin but I think a lot of the treatment of hypertension both with aviators and with regular patients is by force of the physician's personality and the willingness of the amount of time he can bring to bear on the case. One of the reasons I think we should not ground an aviator while we are trying non-pharmacologic modes, is that I believe that hypertension would be diagnosed more frequently if the immediate result was not grounding for a period that may go from four to six months. I think the greatest good for the greatest number is to find as many hypertensives as we can, and I think our current method of immediate grounding keeps a lot of them away from us.

Comment: (German Flight Surgeon): The biggest problem is the compliance of the patient. We need the education, the input of the local flight surgeon with the patient. Everybody in my opinion should have the chance in mild hypertension to, without being grounded, have an adequate trial of non-medical treatment under the close supervision of the flight surgeon. With this approach I think we could have a success rate of 60 to 70% without medical treatment.

Question: Is there any relationship between mild hypertension and increased "G" tolerance?

L/Col Kruyer: I think we have to practice medicine first, and if the pilot has a level of hypertension that needs to be treated, you need to treat it. You could explain to the aviator all you wanted about the risks of hypertension but if you asked him to sign a waiver "will you take the risk by not having your hypertension treated" any F15 pilot would sign it right away. While it may be true that treating their hypertension may reduce their G tolerance, I think you have to be a doctor first. Studies on G tolerance with antihypertensive medications including thiazides and beta-blockers have not shown any significant decrease in G tolerance in normotensive subjects.

Question: How important is the effect of physical training and exercise in controlling hypertension?

L/Col Kruyer: I think that it is an important part of the whole non-pharmacologic approach which included weight reduction, reducing salt intake, stopping smoking, alcohol moderation, and exercise. I think they all go together. Behavioural modification and relaxation therapy are also sometimes useful non-pharmacologuc tools.

Question: Do you have any experience with the results of bio-feedback in aviators?

L/Col Kruyer: I have no statistical data to support its use or to evaluate its use. In a few anecdotal cases I found it to be helpful.

Colonel Hickman: In the USAF, we search for hypertension in various places including the dentist's office. It may not sound fair to have your blood pressure checked just before someone is getting ready to drill your teeth but the fact is that not very many people have hypertension at the dentist's office. Not many people have their blood pressure go up when they see someone in a white coat, or when they get grounded. The truth is that those kinds of people represent a reservoir from which most fixed hypertensives come, and I think they must really be watched very closely.

I think the question about bio-feedback was really an excellent question because there is good documentation in the medical literature, that even patients with WPW syndrome can have heart rate control with bio-feedback, and we just went

through a five year study period in which we demonstrated conclusively that about 80% of our student pilots with air sickness could be desensitized quite effectively as long as we got them back into the airplane quite quickly. We were enormously pleased with our results in bio-feedback treating air sickness in cadets. Once we finished the study, and since we are not in the treatment business at Brooks, we were supposed to turn this treatment modality over to the field. There was rather a stunning lack of interest in the technique in the field. I was really quite surprised at that, and bio-feedback for hypertension also seems to be meeting that kind of resistance.

We are behind in terms of having an aviation psychology service at each of our bases. We are trying to educate our flight surgeons to be more receptive, and I think that if we had educated them better they would be more receptive to bio-feedback.

Comment: (USAF Flight Surgeon). I would like to address that statement. There are really two problems I see in the area. One is that it is not the flight surgeon that is revolting against the prospect of bio-feedback, but the patient, because the bio-feedback centres are all associated with mental health clinics, and no pilot wants to be sent to the mental health clinic for any reason including bio-feedback. They don't want to be seen going to the mental health clinic, they don't want their records to be stamped that they are attending the mental health clinic, they don't want their neighbours to know, and bio-feedback is run by the mental health clinic in the United States Air Force.

The second problem is logistics. We don't have that many bio-feedback centres, and that limits how many people we can send.

Comment: (German Flight Surgeon) We offer autogenic training for pilots, and we have very good success.

Colonel Hickman: One of the problems we face introducing a new hypertensive drug such as capotopril in aviators, is the tremendous logistic task involved. A few years ago when Dr. Hull was at USAFSAM we tried to form a study group of aviators who had failed on thiazide therapy to look at another antihypertensive, and we were not able to find the thirty required to form a study group, so the study had to be done in de novo hypertensives.

In the USAF, in order to study and introduce a new antihypertensive, we have to engage our whole clinical service in what is equivalent to about six solid months of work involving a battery of tests on all members of the study group including psychomotor performance, altitude chamber testing, centrifuge testing, blood volume studies and non-invasive cardiovascular studies. So, it is a major investment for us, but we think we are going to have to do it in terms of one of the ACE inhibitors as the next drug.

The reasons we a looking for a new antihypertensive is not that we have a high failure rate with thiazides. The main reason is the significant number of people treated with thiazides who develop unfavourable lipids or glucose intolerance because of the drug, resulting in a net unfavourable balance in their cardiovascular risk because of the treatment.

DISCUSSION - ATHENS

Dr. Masdrakis: If we discover elevated blood pressure readings in a pilot during the annual examination, we admit them to hospital for more measurements to see if they have high blood pressure. I can't exactly say what is the cut-off for therapy - probably more than 90 diastlic and 150 systolic. We don't have exact definite numbers. We ground them for a short time, advise them to stop smoking, lose weight and then if the blood pressure is still high, we give a diuretic. We do not use beta-blockers in high performance flyers. The regulations are the same for all Air Force pilots.

For civilian pilots, it's a difficult problem. We use diuretics and beta-blockers, but if additional treament is required, we don't recommend a waiver. Because they are older, the cut-off point is higher - greater than 95 diastolic or 160 systolic.

Comment: (HAF Flight Surgeon) We don't take lightly the decision to treat pilots with antihypertensives. We postpone it and see him again and again. If we decide to treat him, we ground him for a short period - a month or so, until we are sure there are no side-effects from the diuretic.

If we do treat them, I don't think there is any need to ground the pilot, and I'd like to ask you why you ground them, because hypertension may increase the G tolerance. At least, it doesn't harm the flyer on a short term basis. So, why do you need to ground them, if you are not going to give medication or to make examinations that also may interact with the flyer?

L/Colonel Kruyer: All I can answer to that right now is that that's what our rule is. That is what our regulation states. When we make the diagnosis of hypertension, it's not based on one reading. If a blood pressure is elevated, and there is a question of hypertension, then the usual procedure is to get two blood pressures per day for five days. It can still be a problem - do you look at the average or trend?

We had a meeting earlier this year to discuss the problem of hypertension, about changing the rules, and changing the approach once mild hypertension has been diagnosed to allow the aviator to continue flying while he starts non-medication

therapies - stopping smoking, diet, exercise - for two or three months. If he is not controlled, then start the diuretic and ground him, like you do, for a short time to look for blood pressure control and side effects. We haven't made that rule change yet, but we are planning to, and the discussions on this trip will help us.

The way our rules are now, the diuretic gets started early, and it often never gets stopped.

HAF Comment: There is no question that for moderate and severe hypertension treatment is indicated, but for mild hypertension, there is no unamious agreement that treatment is necessary and that everyone will benefit. So, especially for pilots, to extend their career, we could probably with clear conscience postpone the point that we start treatment.

L/Col Kruyer: The cut-off number that we have is relatively low - 140/90. You may have an advantage in not having an "official" number. In our Air Force, because of the size, and the number of aviators, and the administration, it would never work. We don't see hypertension at the School of Aerospace Medicine. The problem is handled at the local base, by the local flight surgeon, by the regulations, and that's one reason there has to be a definite number.

Dr. Masdrakas: With your definite numbers, what is your prevalance of mild hypertension in aviators?

L/Colonel Kruyer: I don't know the numbers, It would be hard to get it.

Colonel Hickman: I believe that it is about 2.4 to 2.6%. It is nowhere near the 15% prevalence in the general U.S. population. In aviators on flying status, it is not a problem of great enormity. We are dealing with a relatively young population, even for full service.

A lot of time and energy has gone into discussing what levels of blood pressure we ought to treat. Most of the studies, which were short-term studies, didn't indicate for mild hypertension that there was much advantage in treating them. I have to balance that against the fact that every bit of epidemiologic data that we know about hypertension indicates that it is a no-threshold phenomenon.

Mortality and morbidity are increased even within the range that we consider to be a normal blood pressure, so almost no threshold for blood pressure could really be argued. 140/90 is chosen because that is the point at which mortality seems to be doubled. I think we spend enormous amounts of time arguing over what is a normal blood pressure and I believe that the argument over what blood pressure needs to be treated basically is obscuring an elephant which is staring us in the face. I would be much more satisfied to not treat a lot of the mild hypertensives if I knew who they were, and if their blood pressures were not so regularly obscured by recording 138/88, which is one of the real difficulties of having a definite number. You feel like the blood pressure of 138/88 is the blood pressure that they were issued along with their uniform, and you see it year after year after year. There is a certain tragedy in regulations regarding blood pressure because not infrequently in the cardiac catherization laboratory, when I see a "normotensive" individual with a dilated aorta and I look back through the chart, they have had years and years of 5 day blood pressure checks and eventually, they were able to get an average of less than 140/90 by someone placing them in a dark room for three hours in a somnolent, recumbent position.

So I believe that if you're going to have a regulatory blood pressure, it ought to be as low as possible, because if you moved it to 150/90, I think there would be just about the same degree of obscuration of blood pressure because the person flies an airplane for a living.

Now in the U.S. Air Force we've made one or two mistakes in perpetuating that trend. One is that when people are identified as hypertensives, we've grounded them during initial pharmacologic management or during non-drug treatment, not because we thought that the mild hypertension was a danger for flying, but largely because the non-pharmacologic control for blood pressures takes a great deal of will power on the part of the individual, and continuing to fly is not much of an impetus to get the individual to correct what are life-long habit patterns of self -destruction. That philosphy has turned out not to work.

The real reason that we've recommended to our Surgeon General that we not ground people is that by grounding them (and since they don't respond terribly well to non-pharmacologic measures), we've pushed the flight surgeons into giving diuretics to more people sooner than they would need to, in order to get them back on flying status. We would like to stop that trend.

In terms of drugs, we do not need a new first line drug to treat our aviators because of the failure rate of thiazides. We have looked over and over again at the failure rate of thiazides that would warrant investigating a new drug. In fact, if we want to investigate a new drug like captopril, we will not find enough thiazide failures in any given year to form a study group of 30 failures. We will have to take aviators who are not failures to thiazides, and ground them during our investigations.

The problem with thiazides is that we are concerned about the adverse effects on lipids, glucose tolerance and perhaps on total coronary mortality. This is why we would like to investigate captopril.

G/C Hull: Our experience with our drugs has so far perhaps been fortunate. I think the choice of atenolol was based on exisiting published work and was not justified by trial in military personnel. It is a hydrophilic drug with a very narrow dose spectrum. It has been successful, but there is absolutely no move to licence beta-blockers for use in high-performance aircraft.

There is pressure on the civil side to investigate both captopril or enalapril, and calcium channel blockers, but I cannot see that we in the RAF are going to have any need to introduce these drugs in the forseeable future.

Colonel Hickman: One of the biggest objections that we find to the beta-blockers is that the biggest single medical illness that our aviators have which predisposes to sudden incapacitation is coronary artery disease. The people most likely to have coronary artery disease are those who have coronary risks such as hypertension, and the placing of aviators with hypertension on beta-blockers obscures the symptoms and reduces your ability to detect coronary disease.

Question: Do you use the centrifuge to test hypertensive pilots, and to test new anti-hypertensive medications?

L/Col Kruyer: The centrifuge would be an important part of the investigation of a new anti-hypertensive drug. They would also go through a Holter monitor, echo, treadmill, thallium, as well as centrifuge testing. There would be a full battery of neuropsychiatric screening and performance parameters as well.

Question: Why don't you accept beta-blockers for hypertensive pilots in transport aircraft? Do you have any evidence that this kind of medication is not safe?

L/Col Kruyer: Our main worry is treating somebody with a beta-blocker who has coronary artery disease, and masking the symptoms and our ability to detect the disease until it is more advanced.

A categorical waiver in our Air Force can only be given for a condi ion which is related to G stresses.

Question: For civilian pilots, which hypertensive medication would you suggest besides thiazides and beta-blockers? What is your opinion?

L/Col Kruyer: I think I would suggest thiazide diuretics and capotopril, rather than beta-blockers. I would put beta-blockers third, and then I would use atenolol or pindolol.

Another point I might make is how you choose to interpret the literature is up to you; the literature we went over does not show reduced coronary events, but it very clearly does show reduced total events, primarily cerebrovascular events, and hypertensive cardiac disease. Overall, the weight of the evidence does show that you improve the patient's prognosis by treating mild hypertension. It is just not proven that you improve their coronary artery disease prognosis by treating it.

Colonel Hickman: One of the things that we discovered when we tried to find an additional drug besides thiazides is that for those who did not respond or who were failures to thiazides, within three years of trying to start them on another drug, they had other complications that led to the termination of their flying status - they would develop rhythm disturbances, coronary disease, or their hypertension wouldn't be controlled with two drugs. The thiazide diuretics represented a sort of threshold beyond which the game was not worth the candle, beyond which it was not worth the enormous effort which it took to keep people on flying status who didn't respond to thiazides. As we have followed them, that's a pretty good test.

Only about 20% of aviators are adversely effected in a metabolic sense by thiazides, so we are not in great haste to throw away that drug for a large number of people for whom it is effective and apparently safe.

Dr. Masdrakis: Another thing that should be taken into consideration are the other risk factors for coronary disease, in the decision about when to treat borderline hypertension.

Colonel Hickman: We have a lot of NATO countries now who are treating with beta-blockers, based on the assumption that it is OK. We have some objections to beta-blockers, and if we didn't have those objections, we would test the drug. I would like to see some of the people who are using beta-blockers present to the rest of us some neuro-psychiatric testing data, some psychometrics, some tilt-table, some very simple non-invasive studies to show us before and after that we're not sacrificing anything with those drugs. Even with the hydrophilic drugs, there are certain hormonal changes that might take place and have to do with the fight-and-flight response and the internal milieu. I am a little surprised that beta-blockers have become so widely accepted for tanker-bomber-transport use in a lot of countries without a single shred of data to show what we may be giving up.

G/C Hull: There has been some psychologic testing done at Farnborough, but I don't think it could be regarded as a substitute for actual trials on patients.

We don't seem to have addressed the question of physiological methods. There seems to be no doubt at all particularly such measures as weight reduction and alcohol withdrawal and probably a sustained aerobic exercise program will each contribute a very substantial amount to blood pressure reduction. I think the reason these physiologic methods are not successful is not that they are intrinsically unsatisfactory, but simply that they cannot be sustained.

If in fact it were not impracticable to insist on this, I believe that physiologic methods could be shown to be highly effective in the long term.

Question: I have noticed that people with hypertension sometimes present on the echo with hypertrophy, and their ECG still is normal. What is you decision if you have an air force pilot with mild-to-moderate hypertension controlled by diuretic, and the echo shows mild hypertrophy?

L/Colonel Kruyer: A pilot who showed definite hypertrophy on echo would at the present time be grounded, based on the evidence that his hypertension has been severe enough or untreated long enough to have some end-organ damage or effects. If the hypertrophy were shown to resolve, on later echocardiograms, he could be allowed to return to flying duties. We do not have a requirement to screen all hypertensives with an echocardiogram at a local level.

DISCUSSION - COPENHAGEN

Question: If an applicant to the USAF presents with a blood pressure of 145/90, presumably he is washed out from selection. Is he allowed to present himself again sometime down the road if he undertakes weight reduction and exercise, for example, and his blood pressure is normal?

L/Colonel Kruyer: If their blood pressure is elevated when they are applying, if they can get it down and controlled with non-pharmacologic means, they are accepted into training. If it requires going to thiazide diuretic to control, they are not accepted into training.

Dr. Alnaes: We use the same criteria in Norway, that is 140/90 for selection, but we do pay some attention to the pulse rate as well. If somebody has a pressure of 150/90, but has a tachycardia of 110 at the examining office, we do a Holter and if his pulse rate during most of the day is around 60, we don't pay any attention to that blood pressure.

Dr. Eliasch: Apparently you don't have the same standard as the FAA, whose standard is 150/90 and after the age of 50 or 55 you can have 160/95 and that is their new limits. Am I wrong about that?

L/Colonel Kruyer: I know that that's correct and I might as well make this point; we have nothing to do with FAA and civilian flying. Their standards, many of them, are a lot different than ours.

Dr. Eliasch: But isn't that the ICAO standard?

Comment: The ICAO doesn't have any standard, only recommendations.

Dr. Eliasch: Now there are two things as far as measurement is concerned. The question is, "How many physicians know about the need to place the arm 5 centimeters below the sternal notch?", as has been determined from heart catherizations. That may mean something, because there are a lot of patients that have a large chest. The other thing is patient position, because some technicians would take it in the sitting position, while others would take it in the lying position, and this leads to problem in terms of getting the correct level in relationship to the chest. And then the third thing is the width of the cuff.

I just can't see the point in using 140/90 as a cut off point because you have first to define how you measure it. And so I think it would be immaterial if you used 140/90, or up to the FAA standard.

Colonel Hickman: Well assuming that you have got the patient sitting, and once you have got the correct size of cuff, you still have to make a choice. You still have to say, I must, in a no-threshold phenomenom, pick a threshold.

When we are dealing with millions of people, we have to have something that we can deal with. It's tough to have to pick a threshold, but you would agree that you wouldn't want someone to pick 170/105.

Dr. Eliasch: No I wouldn't, but I wouldn't have 140/90 either. I see the need, but you may be awfully wrong, because it depends on your measurements.

Colonel Hickman: Well I think that you ought to make as many attempts as you can to measure the blood pressure. Technological questions aside, it's the number of readings under various conditions that also helps. We are also looking at several ambulatory blood pressure monitoring devices that we have calibrated in a cardiac cath lab to give us a histogram of the blood pressure over 24 hours so that we don't make the decision on a single reading. We are investigating that because the person

who has 70 percent of all his readings over 140/90 is not the same as one who has 5 percent of his readings over 140/90. And the person who has 10 percent of all his readings over 180/105 for example is again not the same. You need to make the diagnosis on preponderant blood pressure.

I don't think that in the USAF that the technique of doing blood pressures is a problem. Our technicians have all been schooled in how to measure blood pressure. The arms are measured, and we have a wide variety of cuff sizes. Blood pressures are taken in the sitting, hanging-arm position which gives virtually the measurements that you were talking about, so I don't think that it is a problem in the measurement of blood pressure.

I think the problem is in the willingness of a lot of people to say that a casual elevation of blood pressure has meaning. We all know from studies that a single casual elevation is not a diagnostic elevation but the great majority of the pool of fixed hyptensives comes from the reservoir of labile hypertensives and therefore one must investigate more thoroughly those who have isolated elevated readings because they may have definite meaning. Nothing is more disconcerting to me than to do a cardiac catherization on a 40 year old and find an enlarged aortic knob and to look back through the chart to find that they have had a constant blood pressure of 138/88 on all their five day blood pressure checks. To me that is the problem, that is underdiagnosis of hypertension in the person who should have the diagnosis made. This is a bigger problem than the problem of the technologic measurement of blood pressure.

Question: But what do you think about the pulse rate?

Colonel Hickman: Well you get the Holter monitor along with the ambulatory blood pressure. To me it sort of cause and effect, and part and parcel the same problem. To me that doesn't explain a lot away. Most people do not have labile hypertension. One of the places that we check blood pressures is the dental clinic. Not everybody in the USAF sees the doctor, especially if they are not flying, but everybody sees the dentist, every six months. Very few people in the dentist's waiting-room have high blood pressure. Of those who have labile hypertension, a great many of them represent the pool from which fixed hypertensives come. There is an art to doing this, but I don't think that the problems are technologic. I think a lot of readings are a lot more helpful.

Question: Is there additional information to be gained from exercise blood pressures?

G/C Hull: We looked at various measures for discriminating hypertensives in a study which I was involved, and in fact the least discriminating measure was the exercise blood pressure. The maximum exercise blood pressure seemed to provide no discrimination amongst the hypertensive versus normotensive population.

Question: Do you feel that during exercise there is a safe systolic blood pressure which should not be exceeded?

G/C Hull: While customarily we stop at 240, I have no doubt if we tested middle-aged men a substantial proportion of athletic men might exceed 240 or even higher.

Dr. Eliasch: I should worry about treating high performance aviators with any drug if they have a pressure of 140/90 and they have reached the age of say 40 years. Because I do believe they do need that pressure to perfuse the brain in circumstances of high G. If you lower the pressure then I believe that their resistance to G force would be less. I think the reverse is true in normal people without high blood pressure because if you look at those aviators who are very well trained, who run marathons, they usually have a low blood pressure in the range of 110/70 to 75, and they have no problems whatsoever with G forces. So in other words, it doesn't hold necessarily that there should be a relationship between blood pressures and G tolerance, but I wonder if you should really treat pressures such as 140/90.

Colonel Hickman: It's not just by luck that we picked thiazides. It is not without information that we started using them in our aviators. We chose the thiazides only after a long and intensive study in both non-high performance and high performance pilots who were treated with thiazides and examined both before they were treated and after they were treated. The investigations included centrifuge tests, altitude chamber rides, blood volume studies, psychometric tests, Holter monitoring and there was no loss of G tolerance in high performance pilots treated with thiazides. One of the reasons in the USAF that we have to be very careful about choosing a drug to treat hypertension is the complicated logistics of clearing a drug for application in aviators. We are interested in captopril but it will take an enormous amount of resources for us to clear captoporil. The thiazides are an extremely well studied group of drugs in aviators, including high performance aviators in every possible operational condition, so we are not worried about the aviation implications of the drug. We think that ethically it's difficult to continue to use thiazides as the first line drug in a male with mild hypertension simply because the advantage of reducing blood pressure may be out-weighed in some patients by the adverse effect on lipids. We feel we need another first line drug, but it will take us several years of intensive research to clear these drugs for aviation.

Dr. Eliasch: Then it would be more efficacious to make a controlled study having one group of 140/90 given thiazides and the others given nothing at all. You could then look at the rate of strokes.

Colonel Hickman: To what end? That has already been done in the clinical community.

Dr. Eliasch: Not in aviators, and with a blood pressure of 140/90.

Colonel Hickman: Well I can assure you that we will not do knowingly a controlled study and not treat hypertensives in this day and time, when the information is so compelling about everything except myocardial infarction.

Dr. Bonde-Peterson: If you use static/isometric exercise rather than the treadmill, there is an increase in both systolic and diastolic pressure. Recent investigations in children have shown that children with an inherited pre-disposition to high blood pressure increased their blood pressure more during static/isometric exercise than a control group. If you are talking about cerebrovascular insults during the straining manoeuver, then those who actually increase their blood pressure most might be predicted by such a test for hypertension. You might also select out those who increase their blood pressure to very high values. Has there been any consideration to using high blood pressure during straining manoeuvers in screening?

L/Colonel Kruyer: Not as far as a predictor of hypertension.

Dr. Alnaes: Let me ask the converse. Is there a lower level of blood pressure which should be considered as a cut off in the selection of high performance aviators? We would not permit anybody to fly high performance jets if his systolic pressure is below 100. We feel that he wouldn't mobolize quickly enough. We just had a candidate like that who admitted a lot of orthostatic hypotension. Do you check for orthostatic changes?

L/Colonel Kruyer: We don't have a lower cut off point. If they have a problem, they get weeded out because of their intolerance to G. All our TAC flyers undergo G training as well in the centrifuge. I am not aware of us having any problems that might have generated a discussion for a low threshold cut off in relationship to blood pressure.

Dr. Bonde-Peterson: A recent study has shown that manual labourers may show multiple small scars in their myocardium, while this was not the case in a control group. Is there any such evidence in air combat pilots? Could this be the reason for some of the bundle branch blocks?

Colonel Hickman: I don't think that the data exists at present to answer that question in aviators. I have been very concerned that there is virtually cavity obliteration in some cases under high +G, where the walls of the endocardium may actually be rubbing against each other. I think that we will have to see whether there is an excess prevalance prospectively of a lot of corditions that could logically be explained by insults which did not result in gross infarction. The miniature swine seem to be particularly susceptible to these subendocardial hemmorhages, but we plan to start studying primates in our centrifuge, to look at the effects of long term repetitive exposure in those animals. I don't think that the data exists today, but I think it definitely has to be looked at.

DISCUSSION - TURKEY

Colonel Dengiz: Do you have different blood pressure limits for different ages?

L/Colonel Kruyer: No, our population is relatively young, and our standard is 140/90 throughout our population. What are your limits, and what medications do you allow?

Colonel Dengiz: Our cut-off is 150/90, and we do not allow any antihypertensive medications

CORONARY RISK FACTORS IN AEROSPACE MEDICINE

Colonel J.R. Hickman

DISCUSSION - FURSTENFELDBRUCK

Question: If you can significantly increase your HDL, do you think you can reverse the process of atherosclerosis.

Colonel Hickman: When someone comes to a national scientific meeting, and shows me two set of angiograms with regression of coronary disease, I'll believe it can be done. I already believe it can be done in the legs, because I have seen the angiograms. I believe that before the lesions become hard and calcified, that in the legs we have shown regression. I have not seen a case in the coronary arteries that yet convinces me although I have seen two sets of angiograms on a USAF General who started himself on a course of fish oil while on exchange in Canada over a period of two years and it is the first case that I have seen where I think there may be regression. There is not enough evidence epidemilogically, because HDL has only been around since 1979, and that is simply not enough time from an epidemologic standpoint.

Question: What is the reliability of the cholesterol and HDL determinations?

Colonel Hickman: The question about reliability of cholesterol determinations may be the biggest single problem in coronary prevention today. If we are going to place so much reliance on cholesterol determinations, reliance that may lead to invasive studies such as angiograms and advice about changing lifestyles, and if we are going to use these data to determine whether or not a young fellow may be fit for aircrew selection, then the studies ought to be done with maximum precision and accuracy.

Unfortunately, in the US Air Force we just found out that we have been building on sand. The co-efficient of variation for HDLs in particular was abysmal. So, we are solving the problem in one central office. We are directing every Air Force laboratory to utilize a single method, using a "gold standard" from CDC, the Centre for Disease Control.

Currently the College of Americal Pathologists in the US, which does our quality control for all US labs that are certified, *mails a sample and says* "send us back the results of these thiry tests". If you get twenty-seven of the tests right by their standards and three of them are wrong you never hear about it. Furthermore, their "correct answer" is not the "gold standard". Their correct answer is the mean value from the laboratories including those who get the "wrong" answers. So, we want to start our own system with an absoloute gold standard. We want everybody to do it the same way.

Now there is individual variability in total in HDL cholesterol metabolism, but overall most of the variability is at the laboratory and in the test tube and not the patient. I think we are all going to have to attack that problem very vigorously.

Question: Do you think psycho-social stress is a risk factor?

Colonel Hickman: Well, if you look at the person who grew up in Hawaii after emigrating from Japan, and if you look at twin pairs, those who kept the same familial dietary pattern, but adopted a more western lifestyle had a higher incidence of coronary disease. I believe there is something to it. I believe it may be mediated through cortisol and elevated cholesterol.

DISCUSSIONS - COPENHAGEN

Dr. Eliasch: You know there is a lot of enthusiasm for the idea of reducing lipids in preventing heart disease, but you have to realize that there is a lot of resentment as well. It has been shown that by using lipid lowering agents that you can reduce mortality, but it has never been shown that by reducing the cholesterol level approaching the normal range, that you can thereby decrease the incidence of coronary heart disease.

Colonel Hickman: There is a problem though. The normal range as defined by most laboratories is in the range where the disease is epidemic.

Dr. Eliasch: There were so many errors found in the Finnish study, as in all epidemiologic studies. If you are against the idea there is a lot of criticism, but if you are for the idea there is a lot of enthusiasm. I think it is good to do these intervention studies, but I wouldn't dare tell a patient that it has been proven, but just probable.

Colonel Hickman: Well the only way to definitely prove the hypothesis would be to rear two groups of otherwise matched children which is unlikely to happen. The criticisms of the Finnish study were somewhat answered by the cross-over nature. The populations were not migratory, as was a problem in the Framingham study. The diet was controlled fairly rigidly and the cross-over nature of the study made for fairly compelling evidence. I recognize that there are some errors that you can find in any study, but I don't think that it is fair to pick out a small sub-set from any study after it is completed and use that as evidence. The hypotheses have to be set down before the trial begins.

CARDIOPULMONARY SCREENING FOR HIGH PERFORMANCE FLYING: SELECTION AND RETENTION ISSUES

Dr. G.W. Gray

DISCUSSION - FURSTENFELDBRUCK

Colonel Hickman: I think you can't over-emphasize the point that in screening aircrew candidates, you don't want to get into the trap of doing exercise stress tests and Holters across the board, in people who are twenty-one years of age, because what will happen is that you will get a wealth of findings, all sorts of bizarre findings, that are largely due to high degrees of vagal tone. The significance of every finding depends on whether or not there is underlying organic heart disease. So, if you look for those non-specific findings first, you will then have to work backwards to look for structural disease. It is much more cost effective and simpler to look for structural disease first.

I think the answers to the screening problems in aircrew candidates is not going to come from the US Air Force. I think what we have to offer is that we have a centralized facility for aviators who get grounded. We are not centralized for screening aircrew candidates. What we have to contribute are long-term studies in aviators who turn up with problems after they are trained. The Air Forces who do have centralized screening facilities and screen all of their candidates at one place are the Air Forces from whom data on aircrew candidates is going to come, and we in the US Air Force are going to be watching very closely to see what we can learn.

Comment. (Belguim). We have been doing echocardiograms in Belguim for five years on each candidate. We have difficulties in reading the studies. We have the same echocardiogram read by two cardiologists with two different results frequently, even on such straight forward things as measurements and diameters.

Colonel Hickman: I don't think we should let the problem of interobserver variability overwhelm us, because even if we set very liberal standards and not turn down a lot of people based on the echo, we will be so much better off than before we were doing it. The very gross cases that we used to let in and not discover until after they were trained, will be picked up on the screening echo. The Aeromedical Panel of AGARD is going to sponsor a NATO Echocardiographic Working Group both to try to standardize how we do the echos and how we read them, and also to have contributions from every country that does echos in a long-term data bank to look at what the acquired abnormalities are in aviators. I think we will learn a good deal about how to manage the echo, how to read it, how to standardize it, because I could forsee that before the data will be usable for us, those echos will have to be read finally by a reading panel of four or five different countries and almost scored like they do in Olympic figure skating. But I think we will be so much better off screening than not screening, that the problems should not overwhelm us.

DISCUSSION OF CASES PRESENTED BY STAFF COURSE

CASE JP Mitral Valve Prolapse with SVT

JP was a 42 year old KC-135 command pilot in whom mitral valve prolapse had been diagnosed five years earlier based on clinical and echocardiographic findings. On his third evaluation at USAFSAM, during the recovery period following a maximal treadmill test, he had an asymptomatic 13 beat run of supraventricular tachycardia at a rate of over 200 bpm. He was disqualified from flying duties because of mitral valve prolapse complicated by tachycardia.

FURSTENFELDBRUCK

Question: Just to be the devil's advocate, do you really think that because he had that short burst of SVT near maximum heart rate on a maximum exercise stress test, that as a transport pilot he needs to be taken off flying duties for ever?

L/Colonel Kruyer: I don't think I could prove to you yes or no but we believe so, yes.

Going back to a comment this morning about high performance aircraft versus non-high-performance aircraft, we don't look at a multiplace aircraft as having a pilot and a co-pilot, one of whom is redundant. Everybody in the aircraft has duties to perform at a particular time and you may compromise the overall performance of duties if you regard the co-pilot as someone who can take over if something occurs.

Is it worth looking for disease in someone in a multiplace aircraft? We regard all problems except those related specifically to "G", as equally important whether it is high-performance aircraft or not. If you look from the position of Strategic Air Command (SAC) and Military Airlift Command (MAC), if you are going to waiver conditions for a multiplace aircraft because you feel the presence of multiple pilots make it somehow safer, then you make SAC and MAC the repository for your medical cast-aways from fighter jets. If you continue to approach the problem that way, you may well end up with the situation with someone with minimal coronary disease in one seat and someone with mitral valve prolpase and SVT in the other seat.

With mitral valve prolpase, we are talking about a disease with propensity for arrhythmia, and the propensity is always there. We look at it only every three years and so if we find any arrhythmia at all in the three-yearly screen on one twenty-four Holter monitor or one exercise test I think that we have to assume that there may be other episodes as well.

Colonel Hickman: I think if you want to put someone back flying who has mitral valve prolapse and supraventricular tachycardia, you are betting against two things. You are either betting that the two are unrelated, or you are betting that they are related and that that is as bad as it is going to get. The first one is a very bad bet, because it is significant at the .005 level. He may not be one of the ones, but the numbers are so powerful that you must say, "I believe that these two are related". Now they may not be, but the numbers suggest very strongly that they are.

Then, if you give them a waiver, you are saying that you believe that this will not get any worse. I think that these kinds of cases need to be observed in the grounded state in a study group and if it turns out that it is not nearly as bad as we thought, then we can make a case for a waiver. The thing that bothers me most about the study group of mitral valve prolapse from the USAFSAM is that you have an arrhythmic group with CNS events. I am concerned that a significant number of the cerebral events are due to small emboli as they go in and out of these arrhythmias. There may be others who have emboli from the arterial side, unrelated to the arrhythmias. I know it sounds very Draconian to ground somebody for ten beats of SVT and prolapse. It really sounds tough but the thing we have to do is to have a plan to get smart. I think myself it is more liberal than I could bring myself to be, to return someone to flying with SVT and prolapse, although it seems very harsh.

Question: On returning such an individual to flying in transport or other multiplace aircraft, are we not saying that he will not be under such a high degree of physical stress as in a high performance fighter, and then therefore is unlikely to have SVT since the episode occurred only during strenuous exercise?

Colonel Hickmam: The last two strokes that happended in aircrew in the USAF and Canadian Forces happened at rest. They were both people with MVP and arrhythmias, and the embolic strokes occurred at rest. You have a certain degree of leeway with your categorical flying system that we don't have. Our system specifies that no risk can be accepted on the flight deck of a multiplace airplane. You can only put someone in multiplace aircraft if you feel their medical problem is specifically related to "G". You have leeway that we don't have, and if we tried for a system like yours, we would not have categorical flying, because the commanders of our transport and bomber commanders would never agree to it. They don't feel they should take anyone with an increased risk. They feel very strongly that everybody in their airplanes has a full-time job and that they can't afford to be incapacitated.

Now when it comes to small percentages and you say "How worried should we get about these small probabilites"?, we are at the point now in aerospace safety, both from the standpoint of airframes, engines and people that we are dealing with small percentages. If you want to make any impact on aerospace safety today, you are going to have to make it in events that are somewhat remote and improbable because most aircraft accidents today are the result of a coalescing of factors which are rare and improbable but which sometimes come together at one point in time to produce a disaster. The philosophy now has to be in dealing with rare events.

ATHENS

General Psimenos: We are now in the process of selecting our pilots for the Mirage 2000 and F16, and of course the selection is done amoung the existing young fighter pilots, so we have to screen them very meticulously for several things...their spines, vision and of course for mitral valve prolapse, and if this is found even with no other side-effect or symptom, we feel that we have to select them out from high performance aircraft.

Colonel Hickman: I think that is a very wise decision. If we find uncomplicated prolapse in a trained pilot, right now we are returning them to the aircraft that they are already in, but I wish we had the option of moving them to another kind of aircraft. If we could screen them when they are getting ready to upgrade into fighters, it would be the best possible decision. We have very actively opposed anyone upgrading to the F15 or F16 with prolapse. Our real goal is to screen them with echo before they learn to fly.

Question: How many times must you examine a patient with mitral valve prolpase to detect an arrhythmia? How many Holters, etc.

Colonel Hickman: I do not have the answer but I will tell you one thing that we have learned. The mean follow-up on our mitral valve prolapse group is now about six years. We did an analysis of the disqualification rate on sequential evaluations. We had hoped that in the first round of evaluations, we would find most of the arrhythmias, but unfortunately the rate of disqualification is staying exactly even. So, based on one normal exam, you cannot make any conclusion about the future. This is another reason that we have decided we must screen them out before pilot training, because once they get on active duty, it means that we have to keep following them forever with Holters because the rate of productive Holters continues to be stable.

Question: If this pilot with prolapse and SVT was a co-pilot in a civilian airplane, would you permit him to fly?

Colonel Hickman: I would not. I believe the presence of supraventricular tachycardia and mitral valve prolapse is a potentially malignant combination, and I would not allow him to fly. The thing that concerns me most is that we have a population with an increased prevalence of retinal emboli, cerebral emboli and a population that is very arrhythmic, and I believe that there may be a relationship between these periodic rhythm disturbances and embolic phenomena. I'm concerned that they don't faint from their supraventricular tachycardia, and I'm more concerned that they're having repeated bouts of SVT and related embolic phenomena. I feel that for civil aviation, especially for an air transport rating, we cannot overlook this. What we have stated up front is that we will forgive almost any rhythm disturbance unless it is associated with organic heart disease. And then we find a rhythm disturbance and we have organic heart disease, I think we have to say that we have found what we are looking for. We put SVT and VT back in the air, if there is no organic heart disease, because we believe in what we are saying. But, if you put somebody back up with SVT, you have to make the assumption that it will happen again. If you are betting that it won't happen again, you are betting unwisely. So every time you give them a waiver, you accept that this could happen again. If they also have prolapse, I'm afraid I'd have to be pretty strict in that regard, because the organic basis for a recurrent arrhythmia exists, and you have no idea how severe the next arrhythmia may be. Arrhythmias which have occurred in the absence of any organic lesion or electrophysiologic abnormality are usually not so capricious.

I think our FAA, if they were presented with prolapse and 13 beats of supraventricular tachycardia, would say they'd have to see more. The problem is that they give the waiver, and then not look for more.

Eskisehir

Question: What is the Turkish Air Force disposition of mitral valve prolapse. How do you screen your candidates?

Colonel Dengiz: We disqualify all cases of MVP from flying duties. All our aircrew applicants are examined by internists or cardiologists. We have a team of physicians who go to several locations to carry out the initial medicals. Our F16 pilots are specially screened before undertaking training, including an echocardiogram.

Case SR Single Episode of Atrial Fibrillation

L/Colonel SR was a 41 year old fighter pilot with a single episode of atrial fibrillation which lasted several hours. No structural cardiac disease was found, and no evidence of coronary artery disease on non-invasive testing.

Furstenfeldbruck

Question: How did you arrive at the grounding period of six months? This frequently causes a problem with operations, because the operators don't want to change their rules which require check rides and upgrades to maintain currency.

Colonel Hickman: The six month restriction is purely empirical. If the investigation is going to require an invasive study such as angiography before returning the pilot to flying, the longer you wait for a recurrence (which would make the invasive study superfluous) the more justification you have for doing the invasive study. The USAF for instance would require an angiographic study, which should be done at the end of the six month period, because if there were recurrent episodes of SVT in the interim, the angiogram which is being done for a purely aeromedical reason and not clinical reason, would not be necessary.

I would like to reiterate though that when you put somebody back on flying status with a rhythm disturbance, you have to assume that it is going to happen again. You can't give the waiver on the supposition that since there were no recurrences during the observation period, there will be no recurrences in the future. You have to assume that there will be another, and you have to assure yourself that he did not have hemodynamic compromise during the episode. The etiology doesn't matter; if he got weak and fainted during the episode, the game is over then, because you have to assume that that is what will happen the next time. You have to assume that it will recur and going on that supposition you have to assure yourself that the one disease that he may not be able to tolerate in that situation, that is coronary artery disease, is not present. Because if he gets an episode of SVT at 180 beats per minute, and he is in the middle of the Atlantic ferrying one of your airplanes and he has a 40% coronary lesion, he may be in serious trouble after a couple of hours.

Let me add something about tracings. Nothing is harder than to try to make an aeromedical disposition on a tachyarrhythmia without having the tracings. So if one of your aviators shows up in the emergency room in a civilian hospital, and you learn the next day that he has had a rhythm disturbance, it is very important for you to go to the emergency room of the hospital and to get those tracings. His whole career may stand on the balance, because if it is atrial flutter, and not atrial fibrillation, or PAT, the odds that you will want to return him to flying status are very remote. Flutter is an automatic rhythm disturbance with no gating mechanism at the AV node and there is a possibility that he may go one to one and end up at 300 per minute. You can't guess around about what the rhythm disturbance was. You must have the tracings and get hold of them personally.

Athens

General Psimenos: All our Generals are on flying status but unofficially we have lowered the criteria becuase we know they always fly just for the prestige as second (pilot). In a similiar case, we did ground him, so it depends on the rank, which means it depends on the age.

Colonel Hickman: Our Air Force is very similiar in that regard. In the US Air Force you cannot command unless you are on flying status. This is why the Surgeon General gets paid to make the tough decisions, and they have a different set of rules for General officers, because we have so much invested in what they know. We as physicians are not opposed to that. The only thing that we would like them to know is what they are waivering by going through a complete work-up.

I did want to make a comment about the six month waiting period in supraventricular tachycardia. Back in 1973, the USAF had never put anyone back on flying status with an episode of SVT. A retrospective study seemed to be favourable, and it appeared that the underlying discriminator was going to be organic disease, namely coronary disease. We went to the Surgeon General and we said, "If you will put these supraventricular tachycardias back on flying status, we will prove to you that it does not represent a disease diagnosable by current technology". To do that, everyone had to have coronary angiography and electrophysiologic studies. We have learned now that we don't have to do that many studies, but the reason that we had the six month wait was that invasive studies were required, not for clinical reasons but because they flew airplanes, and it allowed time to look in depth for recurrences before doing the invasive procedures.

So if you have someone with supraventricular tachycardia, and in the process of working him up you find an abnormal thallium, I would wait a reasonable length of time, measured in months and not weeks, in which I would look for a recurrences, because it would be unfortunate to cath and then find out he had multiple recurrence of SVT.

L/Colonel Kruyer: It would be possible that the indications would be there to do a cath for clinical reasons immediately, so long as the aviator understands why the procedure is being done now, and that even if it is normal, he will still have to wait six months for a waiver.

Colonel Hickman: The one thing that an electrophysiologic study does not reassure me about is that it cannot reproduce the sympathetic outflow and the sympathetic stress that aviators are frequently exposed to. It is helpful information, but it's predictive value for recurrence is not that good. It's a superb study when people have bypass tracts that need to be mapped, but it's ability to predict whether a tachycardia will recur is limited. In all of the series that have been done with heart rate monitors during flight, the highest heart rates are during the transition phases of flight, during take-offs and landings, when sympathetic outflow is the highest. What you hope is that you don't have atrial premature beats to set up a reentrant disturbance.

Eskisehir

Dr. Gray: In the Turkish Air Force, would you allow this pilot to fly? Would you require cardiac catherization?

Colonel Dengiz: We would want to do a cath to rule out coronary artery disease. We would observe him for a longer period before returning him to flying.

Case RS Acquired Left Bundle Branch Block

RS was a 35 year old aviator with a newly acquired left bundle branch block.

Furstenfeldbruck

Question: Since you can't follow acquired LBBB non-invasively for coronary artery disease, how frequently do you require angiographic follow-up to see if they are developing coronary artery disease?

Colonel Hickman: In the presence of LBBB, the thallium is useless and the treadmill is useless, and the MUGA is not very sensitive for coronary disease. We are still trying to grapple with the question of how long a normal set of angiograms is valid for. We don't have the answer to that question yet.

Eskisehir

Colonel Dengiz: In Turkey, pilots with left bundle branch block are permanently grounded. The most common cause is coronary artery disease.

Case NS Coronary Artery Disease/Angioplasty

NS was a 42 year old fighter pilot who underwent balloon angioplasty of a 75% proximal lesion in his left anterior descending coronary artery.

Athens

Dr. Masdrakis: We know that in the first six months, about 30% (of angioplasties) re-occlude, and now we have the latest results from Gruentzig in the New England Journal of Medicine in which we learn that six months is not enough, that there is later occlusion as well.

Colonel Hickman: Dr. Gruentzig showed a small, but not negligible recurrence rate even after the first six months.

Dr. Gruentzig, as most of you know, was tragically killed in an a airplane crash in Georgia last year, and this was published after his death. Dr. Gruentzig was learning to fly the Lear jet, and unfortunately he and wis wife were killed in some bad weather.

We had three young Colonels that we returned to non-high performance flying after single lesion LAD angioplasty, and they have all done well. They did not have calcification. However, Dr. Gruentzig did show us that recurrences are very common in the first six months, and yet in the following seven years, you still must be very vigilant. One of the things that Dr. Gruentzig showed us that worried us the most is that if you dilate mild lesions of fifty or so percent, that when these lesions recur they are frequently worse than before you started. We felt that even though we wanted to retain these valuable aviators what we simply could not risk was a recurrence that was worse which would place us in the position of having to do another angioplasty which might put them in the operating room for an LAD lesion that they should not have had to be operated for. We became worried about the ethical implications of Dr. Gruentzig's data, and we have stopped doing angioplasties to return asymptomatic aviators to flying. The three original ones are still on non-high performance flying status. We are hopeful that advances in angioplasty such as laser techniques may give us an intervention that will let us return people to flying. We were sorry to have to discontinue this, but it was not the flying results

that we had that were bothering us. Nor did we have any complications in these three cases. It was the risk for our future patients of having a dissection and an emergency operation, or a recurrence that was worse than the original asymptomatic lesion.

Eskisehir

Colonel Dengiz: With his 75% LAD lesion, in Turkey we would ground him. If he had angioplasty, we would not return him to single seat aircraft.

Case CPD Chronic Obstructive Pulmonary Disease

Major CPD was a 40 year old fighter pliot who was a cigarette smoker and had a mild chronic productive cough. He had evidence of small airways disease on pulmonary function testing, and developed marked hypoxia breathing a gas mixture equivalent to 10,000 feet altitude. He was grounded and stopped smoking. Two years later, his pulmonary function had improved, and he was returned to flying duties.

Furstenfeldbruck

Question: Do you think he has become less prone to aeroatelectasis now than he was back when he was smoking?

Dr. Gray: His airways were objectively less stable while he was smoking than on repeat testing after stopping smoking, and based on this objective assessment he was almost certainly at higher risk of acceleration atelectasis at that time. With his improvement in lung function, he should be less susceptible.

Comment: So we should be much more aggressive in our anti-smoking campaign especially in our pilots.

Dr. Gray: Absolutely. I personally don't think we should select people into flying training who smoke and I think we should be very aggressive about trying to get pilots to stop smoking.

L/Colonel Willms: In the Canadian Forces at the present time we are going the opposite way from the general population in North America with respect to smoking. 52% of the Canadian military population now smoke whereas the Canadian general population is now down to around 28%. Women are passing men in smoking, and cancer of the lung as an end point in women is now more common than cancer of the breast in Canada.

Case JC Pericarditis/Myocarditis

JC was a 39 year old senior transport pilot who developed retrosternal chest pain following a viral illness, with ST segment elevation. MB/CK levels were elevated. Coronary angiograms showed normal coronary arteries, with a small area of dyskinesis. He was thought to have suffered a myocardial infarction, and was removed from flying duties. He was subsequently reviewed at USAF/SAM, and had a normal non-invasive work-up. The previous coronary angiograms were reviewed, and were felt to be normal, including the left ventriculogram

Copenhagen

Dr. Alnaes: We had a case in our Air Force very similiar to this man who was killed about 2 years after he had his episode of myocarditis. We were able to examine his heart in detail and there was no trace whatsoever of any scars.

L/Colonel Kruyer: This man had a history of typical myo-pericarditis with viral symptoms preceeding a typical pericarditis-type pain, and the M-mode echocardiogram showed a significant posterior effusion. The EKG showed a typical evoloution of pericarditis with diffuse ST elevation so he had myo-percarditis, probably viral etiology although viral titers were not drawn. We tried to exclude as much as possible other possible causes like metabolic, neoplastic or autoimmune. Viral myo-pericarditis usually occurs 2 to 4 weeks after a viral type illness and resolves in a few weeks. It often follows a sub-clinical course so they may not have pericardial symptoms with it. It may also follow a fulminent course, or it may resolve and recur later, with congestive cardiomyopathy. We had one young Airman when I was in cardiology at Wilford Hall who died while awaiting cardiac transplantation, so the course can be fulminent and fatal. We dug back through the records at Wilford Hall at that time, and found several other similiar cases, and based on those, made some very strong recommendations that when an Airman Basic develops a viral syndrome, he should be excused from all physical training for a couple of weeks.

A few words about pericarditis. This also occurs after a flu-like illness. Estimates in the literature are that 30 to 40 percent may reoccur one or more times over the next several weeks or even several months. This is felt to be on an immunologic basis. It may resolve, or progress over time to chronic constricitive pericarditis.

So our aeromedical concerns are related to arrythmias in the early phase, and in the later phase the development of congestive cardio-myopathy or constrictive pericarditis. The recurrence in the next several months of pericarditis or myocarditis is also a concern. So there are acute considerations, and long term considerations, both of which have aeromedical significance.

So with an episode of either, we will have the aviator grounded for 6 months. He will be watched for six months for a recurrence. If during this period he has no significant arrhythmias or recurrence and has normal ventricular function by MUGA and echo, and no evidence of constriction (based on clinical examination) then he will be returned to unrestricted flying duties including fighters. No cardiac catherization is necessary unless some other part of the examination comes up abnormal. We will then re-evaluate the individual at 1 to 3 year intervals, looking primarily for the redevelopment of constrictive pericarditis or cardio-myopathy.

On re-evaluation, we would do a careful clinical examination looking for signs of constriction, echocardiography looking at resting left ventricular function and pericardial thickening, an exercise MUGA, Holter monitor looking for arrhythmias and a treadmill stress test looking at functional capacity.

Question: What do you think about the use of steroids to reduce the risk of recurrence?

L/Colonel Kruyer: I think that the data on that point is conflicting even for myocarditis, and the majority of cardiologists now do not recommend it.

Case WS Hypertrophic Cardiomyopathy

WS was a 40 year old F-15 pilot discovered to have hypertrophic cardiomyopathy based on progressively increasing ECG voltages subsequently confirmed on echocardiography.

Furstenfeldbruck

L/Colonel Willms: With his hypertrophic cardiomyopathy, he needs to be grounded because of the increased risk of sudden death. He also requires treatment with a beta-blocker because of his obstruction. He should also have his first degree relatives checked.

L/Colonel Rodig: He could also be treated with a pacemaker. Some European cardiologists are treating such patients with a pacemaker because by pacing from the right ventricle you reduce the obstruction (by creating a left bundle branch block).

L/Colonel Kruyer: Aeromedically, the disposition for HOCM is that it is disqualifying for all duties and entry into flying training. We do not recommend a waiver at all. There is an increased risk of death, syncope, arrhythmias, dyspnoea and chest discomfort. There is a 5% mortality for hypertrophic cardiomyopathy, a 20% incidence of ventricular tachycardia, a 50% incidence of arrhythmias overall and a 5 - 10% incidence of atrial fibrillation.

Case JF Aortic Insufficiency/Bicuspid Aortic Valve

JF was a 23 year old active duty A7 pilot who was found to have an aortic insufficiency due to a bicuspid aortic valve.

Furstenfeldbruck

L/Colonel Kruyer: What is the diagnosis?

L/Colonel Willms: He has aortic insufficiency and a bicuspid aortic valve. The systolic murmur is likely an aortic outflow murmur. His other rumbling diastolic murmur is a function of the aortic insufficiency, an Austin-Flint murmur.

L/Colonel Kruyer: This is correct. The diastolic decrescendo blowing murmur along the left sternal border is aortic insufficiency. The systolic outflow is exactly that, and it is because of the increased outflow volume from the significant aortic insufficiency. It is not a reflection of aortic stenosis but rather of increased flow across the valve due to aortic insufficiency and the increased volume load on the left ventricle. The diastolic rumble at the apex is a murmur of functional mitral stenosis, identified correctly as an Austin-Flint murmur. The aortic regurgitant jet strikes the anterior leaflet of the mitral valve, partially closing the mitral valve resulting in a diastolic rumble.

What would you do with this young A7 pilot who has some pretty hard evidence for significant aortic insufficiency, but on the other hand, is asymptomatic. He went to the limits of our protocol on our exercise test MUGA.

L/Colonel Willms: The problem with AI (aortic insufficiency) is that you have to consider replacing the valve six months before you see any of the signs. You need to replace the aortic valve in aortic insufficiency six months before you know you need to replace it.

Question: What would your aeromedical disposition be?

L/Colonel Willms: He should be grounded.

L/Colonel Kruyer: This young man came back a year later, and in the interim he somehow got the idea that if he exercised his heart would get better. He had been competing in triathalons on a competitive basis, and he won all three that he competed in. He exercised a solid two hours every day training for these triathalons. He came back and went for 24 minutes on the treadmill. His echo was unchanged as was his MUGA and his clinical examination was unchanged. He felt that he had proved beyond the shadow of a doubt that he had to be able to fly something. What would you tell him?

G/C Hull: He would probably be allowed to fly with a A3 with us, restricted to fly with or as a co-pilot in transport type aircraft.

L/Colonel Kruyer: He exceeds our limits for a waiver because of the degree of his aortic insufficiency. He remains grounded with USAF but he is contesting the decision to the Surgeon General's Office.

What would you tell him about his exercise program?

L/Colonel Willms: I would tell him that he is killing himself, or rather killing his ventricle. Once you start to see changes in the ventricle then it is already too late. The left ventricle does not recover.

L/Colonel Kruyer: Certainly any form of isometric exercise should not be allowed. Bicycling is both an isometric and aerobic exercise. You also have to be concerned about the extreme degree of exercise, even though it is mostly aerobic, in this young man. He already has a significant volume load on his ventricle, and it is not the same as the volume load an intensive aerobic exercise puts on his normal ventricle. He is placing that load on his abnormal ventricle.

The Bethesda Conference addressed this question very well. I gave this young man photocopies of the relevant parts, in an attempt to convince him that it wasn't just the Brooks cardiologists, but also the famous cardiologists around the world, who are worried about what he was doing to his left ventricle, so he stopped his exercise. Having that article helped a great deal in getting through to him.

Colonel Hickman: One cannot assume that because a person with AI has a normal exercise tolerance that he is not already in some considerable trouble. At Brooks, virtually 90% of all of the AIs we see are put back on flying status because they usually have minimal AI, and it is compatible with a long flying career. The best exercisers that we have are people with AI. They go the longest on the treadmill of anybody, simply because they have a big end-diastolic volume, and even if they have only a low normal ejection fraction, its 50% of a big volume. If you take a person with a big end-diastolic volume and his ejection fraction is lower than 50%, they could still have a very handsome cardiac output. They may look like perfect exercisers because they start with big end-diastolic volumes, but you could be falsely reassured under the circumstances.

Athens

Colonel Hickman: The advantage of the USAF/SAM exercise protocol, a modified Balke, which we've used since 1973 rather than the Bruce protocol, is that it is at a constant speed of 3.3 mph, and a 5% increase in grade each 3 minutes. In order to exercise our aviators on the Bruce protocol, they eventually get to the point where they are running, which produces a lot of artifact. We find if they maintain a constant speed and we impose more of the workload with a gradient that it's a more efficient exercise test to do, shorter, with a cleaner tracing.

Copenhagen

Question: During straining manoeuvers, with the high pressures generated, could there develop insufficiency through a bicuspid valve and is this sufficient reason for disqualifying such an aviator?

L/Colonel Kruyer: I don't think that we would be able to sell the Surgeon General on grounding bicuspid aortic valves for that reason. Quite a few bicuspid valves will develop aortic insufficiency. Since a significant number of bicuspid valves develop insufficiency, by age 40 or so, it is possible we make that more likely to happen if we let them fly high performance fighters. We haven't carried our concerns about aortic insufficiency to that extent yet, but is is a reasonable possibility. About half of bicuspid valves become stenotic by age 45 or so. We don't allow applicants with bicuspid aortic valves to enter flying training. If they are discovered after they are finished training, we do not disqualify them if they have no evidence of insufficiency. If they have insufficiency, then they can only fly tanker, transport and bomber aircraft.

Case RN Tuberculosis

RN was a 39 year old pilot discovered to have active pulmonary tuberculosis on clinical grounds after following a left apical opacity for several years which appeared to be inactive, healed TB.

Copenhagen

G/C Hull: All his contacts were x-rayed and had tuberculin testing. No discoveries were made. Our experience in following-up contacts in the RAF is that no contacts ever developed tuberculosis. Rather, they developed some sort of immunity.

BCG is generally offered during infancy in the U.K., and again in tuberuclin negative individuals in adolesence.

So far as quadruple therapy goes, I think that this would be a fairly standard regimen these days. The only drug we feel compatible with continued flying duties is Isoniazid, used prophylactically in a case of a converter who has no clinical disease, as in the case in the USAF.

DISCUSSION OF CASES PRESENTED BY HOST COUNTRIES

FEDERAL REPUBLIC OF GERMANY

Case 1/Case VL
presented by Noel Mortier, Med Capt Belgian Air Force

V.L. is a 44 year old Marchetti pilot. His risk for coronary artery disease is increased because of his smoking habit (20 cigarettes per day) and his increased cholesterol:

Total cholesterol	283 mg%
HDL cholesterol	44 mg%
Total/HDL	6.43
USAF Risk Index	10516

During his annual medical examination at the Med C LuM (Brussels), his resting ECG showed, for the first time, anterolateral and high septal ischemia. Moderate exercise (a mini-steptest) increased the ischemia. The patient had no symptoms to indicate coronary artery disease. Because of the ECG signs, he was temporarily disqualified as a pilot.

The patient was transferred to the Military Hospital in Brussels for further evaluation. Because of a QS pattern in aVL and slow R wave progression in the precordial leads, a high lateral and anteroseptal infarction was presumed. The cardiac enzymes were within normal limits. The thallium scintigram were interpreted as showing necrosis and/or ischemia of the anteroseptal wall.

The patient was transferred to the University Hospital St Raphael at Leuven. Coronary angiography revealed a maximum stenosis of 90% in the proximal part of the left anterior descending (proximal to the first diagonal branch) and a 90% lesion in the first diagonal. Both lesions were considered operable. The collateral circulation from the right coronary artery to the anterior descending went across the septum.

The patient underwent a coronary artery bypass operation by means of a left internal mammary anastomosis (side-to-side).

He had a normal recovery and remained asymptomatic. A myocardial scintigram 4 1/2 weeks after the operation was normal.

Two months after the operation the Medical Board considered the patient unfit for pilot.

Following an appeal, five months after the operation the Medical Board declared him qualified for pilot with the following restrictions:

1. Only on Marchetti
2. Presence of an experienced co-pilot required
3. No acrobatic flights with G load
4. Valid for a period of 7 months

The reasons for this decision are the following:

1. asymptomatic patient
2. no myocardial damage because of the normal resting ECG and normal post-operative scintigram
3. use of the internal mammary anastomosis which according to the literature has a high long-term patency rate
4. the patient takes no medication, has stopped smoking and follows a proper diet

The patient has to present himself after 7 months for a cardiovascular examination including a resting ECG and a thallium scintigram

My questions are: "Is it true that the internal mammary artery grafts have a better patency rate than venous grafts". We have one study from the University of Brussels (Leuven) which showed that after six years the patency rate was 90%.

Colonel Hickman: There is no doubt that the internal mammary artery implant is a better operation than a saphenous vein bypass graft to the left anterior descending. He did not have a repeat angiogram before being put back to flying. I would also like to be sure that he had a symptom-limited maximum exercise stress test, because a test carried to 80% of the predicted maximum heart rate would not be satisfactory. We know that thallium scintigraphy may have a false negative rate as high as 20 to 25%, even in people who have myocardial infarctions, so that it is anything but a perfect test and it misses coronary disease. I would want to know if the grafts were truly open because technically it is not the same if you do an end-to side as it is if you do a side-to-side. I would have liked to have seen the LIMA re-injected.

Putting all that aside, the real problem is that the operation is not curative, and coronary disease is a very capricious and progressive disease. I would like to know if his angiograms were completely and perfectly normal beyond the insertion of the graft, with not even intimal roughening. I also would like to see those myself, because it is my impression that the operation is palliative and not curative and we now have the problem of trying to follow someone with atherosclerosis in a flying job.

However, if you were inclined to put someone on flying status with surgically corrected coronary disease, this is almost surely the best kind of candidate to do so. I am sure that we would still not certify him to fly, even in a limited fashion in our Air Force.

Case No. 2

A Case of Brutal Bradycardia During Centrifuge Training
presented by Joseph P Vastesaeger - Med Maj BAF
Med C LuM - Brussels

Our second case is that of a young F16 pilot who had some problems during his centrifuge training in the Netherlands. We would like to have your ideas about how to manage such an incident.

This young pilot was 28 years old and had been an F16 pilot for about four years. He stopped smoking one month prior to the incident and he does not drink alcohol. He is not involved in any sport. His annual medical examinations including ECGs and echocardiograms have always been normal.

He is an ectomorph, height 176 cms., weight 65 kilograms. Clinical examination was without abnormalities. Blood pressure 134/86 pulse rate 60.

He went to Soesterburg as all our pilots do for G training. He was not ill the day he went, but he was rather anxious. Before the first run, to 6G, he was quite nervous with a resting tachycardia (145!).

Before they go in the centrifuge, they have a briefing from a Dutch PTO about the ride in the centifuge, and how do the straining manoeuver, with a clear explanation of the problems and how the run is to be carried out. The explanation is very good, but nevertheless, they still feel stressed and have a resting tachycardia.

After going to 6G without a G suit with a heart rate up to 180 during recovery he felt faint and went suddenly into a bradycardia of 40-50/min with an escape rhythm and ventricular extrasystoles. He was close to a LOC. He recovered from the bradycardia and went back into a normal rhythm. Further training was stopped and he was sent to our medical board.

He returned to Brussels for follow-up examination. The clinical examination was quite normal. Psychological examination was carried out because of the signs of fear and nervousness at the centrifuge but this was quite normal. An echocardiogram was normal. We did a Holter monitor during flight. This was the first time we had received such information from the centrifuge, and a political decision was made that we could not use such information to ground a pilot. So, we did a Holter in flight in an F16, pulling up to 7G. We also did some studies at the Centre, including a tilt-test and exercise test which were normal.

After a period of three months restricted flying with a copilot, he was declared fully qualified and is still an F16 pilot.

Rhythm disturbances are often seen during centrifuge training. In this case the onset of bradycardia was so brutal, at low G, and almost accompanied by a loss-of-consciousness that further training was stopped. The incident was interpreted as a nervous hypervagal reflex due to some degree of anxiety before centrifuge training. This pilot was allowed to fly again but it was recommended that he have more physical training.

What further screening tests should we do? Is he prone to further such incidents? What is the predictive value of rhythm disturbances during centrifuge training?

Colonel Hickman: He appears to be a normal person from all respects but he is somewhat tall and thin. Now I don't work at the centrifuge and I wouldn't want to pass myself off as an expert in acceleration physiology. But post-G bradycardia is a finding so common that if we disqualified fighter pilots for what we see here, we would have a lot of disqualifications that would be hard to explain.

One of the things about subjects with mitral valve prolapse is that during "G" stress, sypmathetic tone tends to dominate and they have a lot of ectopy but parasympathetic tone tends to dominate as soon as the "G" is released. The case still has the smell of mitral valve prolapse to me, even though everything possible has been done. I would examine him with amyl nitrite, both with echo and ausculation to see if I could bring it out. If I found anything that suggested prolapse, I would do an angiogram. But I wouldn't do an angiogram based just on what I see here. If he had scoliosis, a pectus, or if he had a click with amyl, then I would do a left ventricular angiogram to be sure he does not have prolapse. I would not fly someone with prolapse who may be prone to having this again, because with prolapse, the disturbances are not reproducable. I believe you have made the right desicion, I think you have made the best decision possible and it is exactly what most of us would do in this circumstance.

Is this not frequent enough, this post G bradycardia, that we have to live with it somewhat?

Major Van Holten: Yes, we see it frequently, and also we see changing rhythms where you get a bradycardia and then back to a tachycardia and then a bradycardia. We see it especially when the G load is decreased, and that is a different situation than occurs in flight because when you are flying, and you stop pulling G, you are in a one G environment. In the centrifuge, when you stop you still have a G load for a

few seconds so I think the biggest problem with this pilot is that he couldn't get above 6G. The tachycardia of 140 before the start of training is also unusual, although a resting tachycardia of say 100 is not unusual before the start of training.

Colonel Hickman: That's another thing that makes me wonder about accoustically silent mitral valve prolapse. I would do another treadmill test and I would look closely for vaso-regulatory abnormalities, with hyperventiliation changes or standing changes and if I saw those I would be a little bit more suspicious of MVP.

Major Van Holten: Often when there is a high heart rate at the start of training, as the heart rate increases with the G load, so that by 6 or 7 G the rate is up to 200 beats per minute, then the G tolerance and endurance is lower, so they can't sustain training.

Question: What standard are you looking for with G training, that is how many G for how many seconds?

Major Van Holten: 8G for 15 seconds.

Question: In the Belgian Air Force what is your standard or do you have one?

Answer: It is only training. We don't have specific limits for tolerance. We follow the Dutch program.

Major Van Holten: It is good that you stress that point, because we are not using it as an evaluation, but for training.

Colonel Hickman: We are at a bit of a disadvantage here asking someone from the US Air Force about this question because the only aviators who have a monitor on them in our centrifuge are the ones who are actively undergoing a medical evaluation. Before our Tactical Air Force pilots could be trained, we had to come up with an agreement that they would not even receive electrocardiographic monitoring. Our fighter pilots do not have any leads on them when they undergo this training, and we do not know what is happening to them. I believe this is a fairly common phenomenon from our medical evaluations but it is not available from our training pilots.

Question: What proportion of pilots can achieve 8G for 15 seconds?

Answer: Almost all

Major Van Holten: I think this pilot needs a further familiarization ride in the centrifuge. I think this is very important. He then can have more training with and without anti G manoeuvers and then he may tolerate a ride up to 9G. With this pilot, I would feel very comfortable after a break of a couple of months to have him return to the centrifuge and the first time just to sit in it, and then to have some familiarization rides.

USAFE Flight Surgeon: We have been sending both F15 and F16 pilots to Soesterberg for training, and we have had an excellent time working with them. We have so far over three years only one F15 pilot who couldn't complete the entire profile. We put him on an exercise program, alternating aeorbic and isometric activity with recurrent training on how to do the M-1 manoeuver properly. We sent him back to the centrifuge after six months and he passed with no problem.

Something to consider with this pilot would be just confidence. Maybe he needs to be put on a regimented exercise program, with repeated practice with the M-1 manoeuvre over several weeks, in order that he would have no problems. It sounds to me that he was suffering from confidence problems.

Colonel Hickman: You don't mean running four miles a day now do you?

USAFE Flight Surgeon: Well, it doesn't make a lot of difference, Statistically you could run three miles a day and it doesn't matter. Statistically, there is no difference on G tolerance. We have already shown that and the literature is out. I know what TAC says, but here at USAFE we disagree with that.

Case 3 - Coronary Risk Factors
L/Colonel Erich Rodig

During our annual aircrew examinations here at Furstenfeldbruck in 1986, of all pilots examined we found on the resting and bicycle ECG, 182 cases of ventricular ectopic beats (4.7 per cent). The observed 8.7 percent in propeller pilots is likely due to their higher average age.

On the exercise test, subjects were required to reach at least 2.1 watts. per kilogram of body weight and a pulse rate from 130 to 170. In 140 cases we found ventricular ectopy. In looking at the Lown classification, in more than 30% the ventricular ectopic activity was Lown grade 3 to 5.

These pilots underwent a full cardiovascular examination. In the 182 cases we found 21 cases of mitral valve prolapse. These were trained pilots flying fighter planes and they presented a dilemma to us. In 131 cases we didn't find any organic

heart disease. The most common problems were hypertension and mitral valve prolapse. I think there is also some silent ischemia that we aren't detecting.

With this background I would like to introduce this case, a 49 year old cargo aircraft pilot in the German Air Force. This case may be considered an example of the significance of cardiovascular risk factors in the development of coronary artery disease.

For a period of 28 years, that is from 1957 when he joined the Federal Armed Forces, until 1985, when the disease became clinically manifest, this officer who is only 165 cm tall, has been continually overweight (7kg average, 16 kg max). An additional cardiovascular risk factor apart from lack of exercise and slowly developing hypertension due to stress was the the abuse of nicotine, i.e. 25 cigarettes per day over a period of more than 20 years.

Lt Col R. had been without significant clinical findings besides an autonomous thyroid adenoma in 1980. In March 1985, he felt paroxysmal pain in his left arm for several days, accompanied by slight sickness and nausea. An ECG was obtained and found to be normal. In contrast to this, the ECG obtained five months later for military flying duties revealed typical signs of a previous anterior wall myocardial infarction. Coronary angiography confirmed the infarction and revealed coronary disease with medium to high-grade three vessel involvement. An aortocoronary multiple bypass operation appeared indicated, and successful surgery was accomplished in September 1985.

Post-operative recovery was without complications, and subsequent repeated ergometrical performance tests in a rehabilitation center revealed favourable maximum stress-response without any electrocardiographic signs of ischemia or rhythm disturbances.

Nevertheless, the officer's request for a special flight permit by the flight surgeons was declined by the Air Force Institute of Aviation Medicine with reference to the particular nature of the basic disease and the overweight condition which continued to be manifest.

Case 4 - Cardiovascular Disease and Bypass

A military pilot, at that time aged 42, showed discrete signs of disturbed repolarization on a stress EKG in 1975. One year later diastolic work-load hypertension was found. There were no changes in the patient's condition until 1983. On his annual check-up in 1984 the stress EKG showed a significant repolarization disturbance for the first time. Cardiovascular disease could not be excluded by thallium scintigraphy. Coronary angiography showed severe three-vessel disease. There were no signs of myocardial infarction.

A bypass operation seemed indicated and was done in January 1985. Eight months later heart function tests showed excellent results. (PWC 150: 2.9 W/Kg at 225 Watts). Heart rhythym and blood pressure were not found pathological.

The pilot was permanently refused his licence.

L/Colonel Kruyer: I would not let this pilot or any other pilot post-bypass surgery fly in the air force for a number of reasons. Dr. Hickman quoted a significant yearly closure rate for bypass grafts, and in this case the grafts are all saphenous vein grafts. If he had had any internal mammary artery implants, it would have only been to the anterior-descending or diagonal branch, and he would still have the saphenous grafts to his circumflex and his right coronary artery, so those would still be at that same significant closure rate. This gentleman's native coronary arteries are going to occlude within a relatively short period of time. He has more than one significant lesion in all three coronaries, and his native coronaries will occlude, probably within a year. He will then become completely graft dependant, so that when he closes the grafts, unless he somewhere develops collaterals, you can almost guarantee that he is going to have an infarct. He is also looking, depending on how long his grafts stay open, at a possible second surgery.

Even if he had a completely normal postop. thallium, treadmill and MUGA, there is absoloutely no way that you could predict a future event. There are definite statistics in the literature that he has potential for big trouble, and definite knowledge that when that trouble occurs it may be extremely abrupt, i.e. sudden death or infarct.

Colonel Hickman: All of our aviators who come to USAFSAM as part of their normal work-up get a KUB film, and we see a reasonable amount of iliac and femoral calcification. We did a series of ankle pressures, looking for significant hemodynamic impairment. We don't think that the search for differential pressures in the legs during treadmill tests is worth the effort in trying to find vascular disease, but the KUB film in looking for iliac and femoral calcification is worthwhile. I don't know if it is good enough that we will ever go straight to catheterization, but the finding certainly raises the probability of there being significant coronary artery disease.

We also do coronary fluroscopy on our patients. This patient had a little calcium in his LAD. I think when you are concerned about coronary artery disease, no matter what the other non-invasive studies show, I would do coronary fluroscopy because calcification in a coronary, we have found, is an extremely important finding, and we pick up significant numbers of definite coronary disease, with normal exercise tests and normal thalliums, by finding calcification on fluroscopy.

L/Colonel Kruyer: I think you said that the internist, because he was asymptomatic, was not in favour of surgery. In his pre-operative state, the most bothersome things about this patient is that he did not have warning angina. He has ischemia by treadmill, by thallium, and by MUGA and then came in and said he had run 3,000 metres a couple of days before with no symptoms. I think we have to be very concerned about what is happening to this man when he is running. We know he is ischemic, and it is very bothersome that he has no warning system. It would be much more comforting if you were considering not operating, if he did have angina. With medical treatment, there is no way of judging its effectiveness since there are no symptoms. This is not an unusual situation to see in aviators, that is, patients with no symptoms in the face of significant ischemia with exercise.

Colonel Hickman: Not everybody with asymptomatic disease needs to be considered for an operation. But if someone tells you that the coronary artery surgery study (CASS) showed that the patients operated on didn't do any better than the patients who were treated medically (the European Heart Study showed that in a couple of sub-groups the surgical patients did better), then you should note that in the CASS study, over 22,000 patients were considered before they were enrolled, and less than 1 out of 8 of them entered the study. Everyone of the patients who entered the CASS study had stable angina. The people who dropped dead were never considered for the bypass surgery obviously. The people who presented with unstable angina neither were considered. The CASS study was on people who survived their first symptom, that is, angina. In this case, what this man says is,"Do you think I will be one of those lucky enough to present with stable angina and be operated on and do as well as those in the CASS study?"

No one knows exactly what is right in this case except that it is a deadly situation to have no warning. All that you can conclude from the CASS study is that bypass surgery is a highly effective operation for the relief of symptoms, and that in most individuals with stable angina, the operation can be postponed for a considerable period of time safely. Don't let anyone not allow a patient like this to not be considered for surgery by quoting the CASS study. It is not applicable.

Case 5 - Recurrent Pericardial Effusion

This 38 year old helicopter pilot had a traffic accident in 1981 and suffered a contusion of the thorax. Within two weeks he developed increasing respiratory distress and pressure sensations in his chest. On admission to the University Hospital of Marburg he was in shock, caused by a hemopericardium. He was discharged four weeks later with complete restoration of health within the following year.

In 1986 he was again admitted to the hospital in shock, due to another pericardial effusion, now caused by a toxoplasmosis infection. After therapy, he got a full recovery .

The problem is, what caused the first effusion? Was it traumatic or was it toxoplasmosis? Until now, we guessed that the first one was traumatic and the second one was toxoplasmosis.

However, toxoplasmosis gives a lifelong immunity after the initial infection and after the second episode of hemopericardium due to infection with toxoplasmosis we could see an acute phase antibody and this has been going down. I think it is clear that the second episode was an acute toxoplasmosis infection. Since this was a toxoplasmosis infection, he couldn't have had one before so the first one was traumatic.

Colonel Hickman: I wonder if having such injury before makes you more vulnerable to having an infection later. I would only be guessing there. Was consideration given to a pericardial window after he had the second effusion instead of doing a second pericardiocentesis?

L/Col. Rodig: No, but the 2D echocardiogram is normal now. There are no rubs or mumurs.

Question: Between the first and second episodes, did this effusion completely resolve?

Answer: Yes, there were several echos between the two episodes and the effusion was completely gone.

Colonel Hickman: I think aeromedically you made a good decision. I think he is a good candidate to keep on flying. We have had a few cases of myocardial contusions and we have managed those almost the same as we do with pericarditis. We did have a case in which there was herniation through the pericardium. It was not operated on and he is doing well but we never felt that we could put him in a position of potential rapid deceleration. We did have a few cases that we picked up where there was congenital herniation through the pericardium and we removed them from flying, but we have never seen anything like this before. Most of our cases of pericarditis have been of the usual viral type. I would urge you to publish this case, because I have learned something about toxoplasmosis.

Case 6 - WPW Syndrome

HD is now a 28 year old pilot, flying Do-28 airplanes. During a regular check-up in 1984 his exercise ECG demonstrated intermittent (1:1) Wolff-Parkinson-White conduction pattern and some ventricular premature beats. HD was disqualified from flying training.

A 24 hour ambulatory electrocardiogram showed varying PR intervals, but no paroxysmal tachycardia, atrial fibrillation, supraventricular or ventricular premature beats.

In 1985 invasive eletrophysiologic testing was performed in the University Hospital of Hamburg. A long refractory period of the bypass bundle and the AV node was found. By rapid atrial stimulation no tachycardia or atrial fibrillation could be induced. The bypass bundle was blocked promptly after the intravenous infusion of propafenone.

Echocardiography showed no structural abnormality, especially no manifestation or forme fruste of Ebstein's anomaly.

The resting ECG demonstrated intermittent WPW conduction pattern in April 1985 and complete bypass conduction later in November.

Up to this date, there is no history of heart sensations, tachycardia, lightheadedness, syncope or presyncope. HD is in good cardiovascular condition.

There are some agruments that there is no increased risk for tachycardia or syncope:

- invasive electrophysiologic testing
- one bypass bundle with long refractory period
- no tachycardia or atrial fibrillation could be induced by rapid atrial stimulation
- echocardiography : no structural abnormalities
- Holter ECG - no tachycardia, atrial fibrillation
- proband's history: no tachycardia, no syncope, no lightheadedness

After these diagnostic measures the pilot got a special waiver, excluding him from flying jet airplanes.

L/Col. Rodig: We made the decision to waiver him in the DO-28, a 2 seater aircraft. We had a long discussion about him after we had the results of the EPS and stimulation studies, with no tachyarrhythmias, and we made the decision to allow him to fly. I don't know if you agree?

Colonel Hickman: We do not take WPW EKG finding into flying training because we believe there is an increased risk for tachycardia. There are also other practical reasons that we don't. We have for a long time been denying these people into flying training. When the Fourth Year students at the Air Force Academy are getting ready to go flying and have their cardiograms done, some of them who were not conducting down their bypass tract earlier when they had their cardiograms done, will now be going down their bypass tract. There are a number of them every year, in which this happens.

We recently had the son of an airline pilot in such a situation and the USAF turned this young man down. So he went to a university electrophysiologic department and they did a very extensive three day electrophysiologic study on him. They used four catheters and did all sorts of drug studies, and they proved beyond a shadow of a doubt that his bypass tract would fatigue antegrade at a heart rate that was very low and would not conduct retrograde at all. So he did not in the very true sense of the word have ventricular pre-excitation. He had only a vestigial bypass tract.

For a University that is one interesting case. But if we get into the business of doing electrophysiologic and other invasive studies on young men who have not yet learned to fly, that would greatly change the way we do business in the US Air Force. For us, we would have to resort to doing this a significant number of times a year and I am sure that a University centre would also tire of doing normal people. It doesn't matter if one applicant has a vestigial bypass tract or they all have it, you still have to stimulate them all once you've made the decision to do so.

We reserve invasive procedures for people who are already trained to fly. We do not do invasive procedures on the people who only want the opportunity to learn a dangerous occupation. There are other ways they can serve their country. They can still fly civilian aircraft. We don't even know if he has the right composite of psychomotor skills to become a pilot. We don't know if he will be one of the significant percentage who fail and don't make it through pilot training. We know the odds are very high that he will only fly for five years for the military. We have no guarantee that he will be a 20 year air force aviator. All of this makes us very reluctant to do dangerous tests on people for the opportunity to learn to fly, but if someone presents us with a case like this we will accept him for flying training. It is just that we refuse to do invasive studies because the numbers would become prohibitive.

Now the other problem is that if we took WPWs into flying training, within ten years we would have 200 of these flying in our air force with the kind of medical surveillance that they require including repeated Holters and other studies. I don't think you should take people up front into flying training who you already know require more medical surveillance than everyone else. While he may be able to tolerate a tachycardia of 180 beats per minute when he is 22 years of age, the one disease that he is most likely to acquire as an air force pilot is coronary artery disease and at age 38, with a 70% circumflex lesion he may not tolerate that

tachycardia and yet the treadmill will be totally useless in WPW because it will virtually always be abnormal. So then for his cardiac surveillance we are into thallium scans. It is a logistical problem that we are not willing to tackle, just to give someone the opportunity to learn to fly.

One thing that we have to be careful about is that when we screen candidates we must do it in the most cost effective way. Most of the ones we fail are because of their eyes. We are not going to do echocardiograms as the first step because the eyeballs will flunk out more people. Now if someone is going to have an invasive procedure before he starts flying training it would be a good idea to know that he passes all the other required tests. We have had people go out and have their eyeballs fixed with radial keratotomy in the hope that they may fly not knowing that they had mitral valve prolapse. If people are going to go out and have procedures like this, they should get a very thorough work-up of all the other possibilities because the procedures may come to nothing if some other problem is discovered.

In aviators who are experienced and who are already trained to fly we would not do an EPS. We would accept the fact that he has a bypass tract. We do our first cardiogram after entry at age 35. So if he went into pilot training and we didn't know he had a bypass tract and we found it at age 35, we have 14 years without tachycardia by history and the odds of having an episode of tachyarrhythmia go down significantly. We do a full work-up but we do not do a technical EPS because we already know he has a bypass tract and we suspect that we could probably stimulate a significant number of these into PAT. But we accept them as they are and we don't restrict them and let them fly everything. But that is because we don't find it until age 35.

Dr. Gray: We have candidates like this as well and we are currently dealing with one who has completed primary flying school and has an intermittent WPW. We have disqualified him from going on into jet training. He has, on his own, indicated that he will go and have his bypass tract sectioned and I wonder what your comments are on this approach? Would you accept him if he has this done?

Colonel Hickman: Well he is in a real bind there because cardiac surgery is disqualifying from flying training and we would find it very difficult to waiver him under those circumstances. I guess if he had a perfect result it would be hard to deny him. The thing I worry about is for him to take the risk of having an operation and the possibility of developing a complete heart block and having a pacemaker for the rest of his life by being operated on when he doesn't need to. Someone needs to talk him out of this because even with the best electrophysiologist in the world and the best electrophysiologic surgeon either with cryoablation or with the knife, he can end up with a complete heart block and a pacemaker at age 20.

CASE DISCUSSIONS - GREECE

CASE 1 : OBSTRUCTIVE PULMONARY DISEASE

Presented by Dr. Giatromanolakis

Patient : Mr. T.T., age 57, Olympic Airways airline pilot

This airline pilot's symptoms started in 1975 with seasonal allergic rhinitis, and since 1980 he has been complaining of breathlessness and chest tightness mostly on physical or emotional exertion, during respiratory infection or after inhalation of bronchial irritants. He reported an attack of dyspnoea during flight in 1983. He quit smoking in 1983. His mother and one of his sisters suffer from bronchial asthma.

On periodical examination every six months in the HAF Aeromedical Center he has been constantly found to have wheezing, mainly expiratory, fluctuating in intensity. The chest x-ray has been normal. FVC 3650 ml (89%) - 3400 (84%) - 2700 (67%). FEV_1 3050 (83%) - 2800 (88%) - 2500 (81%) - 2300 (74%). FEV1/FVC 73% - 70% - 74% - 85%.

There was no improvement after inhaled salbutamol, and expiratory flow rates at 50% and 75% vital capacity were also significantly reduced.

Allergic skin tests give positive reactions to house dust mites and feather mix. He is almost continuously under treatment with oral theophylline (Theodur 300 mg q 12 h), inhaled salbutamol (two puffs q 6 h) and inhaled beclamethasone (two puffs q 6 h). A one month course of oral methylprednisolone had no effect on his spirometry.

The pilot has been temporarily grounded.

Topics for Discussion
- Flying fitness
- Medication and flight
- Allergy and flight in the HAF

Dr. Gray: I am afraid I would have to agree that he is no longer fit for flying duties, and the right decision has been made to ground him. I think it is very unlikely that he will ever be able to go back flying again.

He's obviously got moderately severe obstructive airways disease, and although on pulmonary function testing there was no clear response to inhaled salbutamol, a definite reversible component has been demonstrated by his clinical course of variability in his wheezing, and variation from day to day. This clearly puts him in the category of asthma, although there may be a degree of background fixed airways obstruction.

He has definite and worrisome symptoms of dyspnoea and chest tightness, and he's had at least one episode of reported bronchospasm on the flight deck. He has a number of triggers for his airways disease, and he gets wheezy when he gets anxious, an undesirable combination for flying duties.

I think the medications that he's on would not permit him to fly in Canada as a civilian airline pilot, the Theodur in particular having a narrow therapeutic/toxic margin, with significant side-effects that are incompatible with flying duties.

The only medications we would allow in Canada, as in Britain as Dr. Hull covered yesterday, are inhaled sodium cromoglycate, and inhaled salbutamol. If he were able to be controlled completely on those two medications alone, consideration might be given to allow him to return to flying, but he obviously has more severe disease than can be controlled with those two medications alone.

In the Canadian Forces, as in the RAF as Dr. Hull described, pilots with mild reactive airways disease well controlled with inhaled sodium cromoglycate and/or beclamethasone may be returned to restricted flying duties, with or as copilot, but unfit fighters.

Since he has significant upper airways allergic symptoms as well, there is some possibility that these symptoms might be ameliorated with an antihistamine such as terfenadine. In Canada, we allow pilots to use terfenadine on an as required basis under close supervision of a Flight Surgeon, in other than high performance fighters and tactical helicopter operations.

Unfortunately, I am afraid this chap has finished his flying career, and will require some fairly intensive treatment over the years since he is not completely well controlled on his present regimen.

Dr. Giatromanolakis: He has now accepted the fact that he is not returning to flying.

Dr. Gray: I would like to comment on the use of airway challenge testing, for example with methacholine, in assessing aircrew with reversible airways disease. We use it frequently in young aircrew candidates who have any history of asthma during childhood or who have significant upper airway atopic symptoms, to see if they have objective evidence of increased airway sensitivity.

It's also useful in experienced aircrew who develop mild reversible airway symptomatology to help objectively quantify their airway sensitivity, which may not be fully disclosed on history.

It's less useful in persons with clear asthma such as in this case, and I don't think there would be any helpful information obtained in doing an airway

challenge test here. It is in this kind of asthmatic that it can be a dangerous sort of procedure, in that even rather low doses of methacholine may provoke a serious asthmatic attack. While in young aircrew candidates there is virtually no serious risk, as the individual's airway sensitivity increases, it becomes more of a risky procedure.

Colonel Hickman: What would you recommend for an applicant for flying training who has had no symptoms of asthma, but who has a strong family history of asthma?

Dr. Giatromanolakis: In Greece, if he has no symptoms or signs and his pulmonary function tests are normal, I don't think I have the right to prohibit him from flying training. We would allow him to be a cadet. He may not turn out to be allergic anyway. The methacholine test may be helpful, but we haven't set up this test here in Greece.

Colonel Hickman: Why does someone develop overt clinical asthma at age 45?

Dr. Gray: The mechanism of so-called intrinsic asthma or asthmatic bronchitis which develops in older persons is not entirely clear, but to some extent at least involves increased airway sensitivity resulting from long-term exposure to bronchial irritants, in this particular case, cigarette smoke, which produces the histopathologic changes of chronic bronchitis with goblet cell hyperplasia et cetera, and at the same time increased airway reactivity. These airways with increased sensitivity then react to any of the non-specific airway stimuli including exercise, cold air, respiratory infections, etc. I believe that primary allergic mechanisms play a very minor role if any in this type of problem. The real problem is the long-term exposure to bronchial irritants, which usually turns out to be cigarette smoke.

Colonel Hickman: So it may not be a good idea to choose someone with a strong family history of asthma, who is a smoker. Some airlines do not choose to hire smokers for an airline pilots job. They test applicants with urine nicotines, and they turn down pilots who are smokers.

Thinking from a military standpoint, if you were going to turn down smokers, you should do bronchial provocation testing on people with a strong family history of asthma. The two together may not be a very good investment.

Comment (NAF Flight Surgeon): In Holland, we take a special family history. We ask the mother and father to fill in a form, and often we have a positive finding - eczemza, atopy - that allows us to do the challenge tests.

In Holland, hyposensitization also has a bad name, not with the authorities but with the doctors, because of non-compliance, and anaphylactic reactions. It is also difficult to find a specific allergy and to correlate skin tests with the patient's symptoms.

Dr. Giatromanolakis: A recent article in the British Medical Journal pointed out that it is possible to expect to some benefit with desensitization in grass pollen and insect allergies, but it is debatable for house dust mites. It points out that in Britain, desensitization can be done only in the hospital now.

As far as pilots are concerned, it's a matter of career, so even if I can expect only a small percentage of success, it is still perhaps worth thinking of it, for example if I have a pilot with seasonal rhinitis and asthma, in case I can help even in a small proportion, I would seriously consider this treatment, and I wonder what you opinion is?

Dr. Hull: Regarding this particular case, I agree with Dr. Gray. I really cannot see any way this particular individual could be re-licenced for duties as an airline pilot. His disease is too severe, and the prognosis is too uncertain, and is not good overall.

Of the drugs available, the beclamethasone inhaler is perfectly acceptable (for flying duties). I don't think he's tried cromoglycate, which would also be acceptable. I believe that in civil operations, provided he was a responsible individual who understood that salbutamol was potentially toxic, and provided he's been on this dose for some time, then I think that is probably an accepatable preparation for "as or with copilot". I am very unhappy about theophylline containing preparations. Apart from general measures such as avoidance of allergens, I really don't think there is any other treatment that I would consider acceptable.

Case 2 : Linear lung shadow
Presented by Dr. Giatromanolakis

Patient: Fighter pilot, Major G.T., age 37

Health Problems: Mild unstable hypertension 155/105 mmHg
Overweight: 103 Kgr (Height 1.75 m)
Smoker: 1 pack/day for 20 years

Pilot since 1968 in T-33, T-37, F-84, F-4 Phantom, with 2000 flying hours, breathing 100% 02 occasionally during flight.

Chest problem: Linear shadow in his right base anteriorly, found at his periodic medical examination in 1979, unchanged ever since. He is asymptomatic. Clinical examination normal. Spirometry normal. Fiberoptic bronchoscopy normal. Mantoux test negative.

Topics for discussion:
Pathogenesis of the shadow
Flying fitness significance
Clinical significance

G/C Hull: Regarding the nature of the shadow, we know that it is an abnormal shadow and presumably has been there now for eight years, and so it represents very old pathology. When I looked at the chest x-ray, I wondered whether it was atelectasis at all. The usual rule for atelectasis is that it must radiate from the hilum. However, in this case I think this is probably an atelectasis of either the lateral or medial segment of the middle lobe, and although we don't have a lateral film to prove this I believe we are seeing it on a slight angle on the PA. On the tomogram, the shadow is dense anteriorly. I think we can accept that this is a chronic atelectatic lesion involving one or other or both segments of the middle lobe.

We know he has had a bronchoscopy which has presumably excluded any underlying intrabronchial pathology, and therefore I think this represents old history.

The question of pathogenesis is clearly somewhat speculative. It is possible that he had some G type atelectasis, but I've never seen that result in permanent damage. I think it far more likely that this gentleman, as a long term and heavy cigarette smoker, was prone to pulmonary infections, and he had an infection which resulted in a small area of inflammatory atelectasis.

I don't believe that in view of his normal respiratory function that it has any immediate practical significance as an aeromedical hazard. I think it should be pointed out to him that there is an area of damage in his lungs already, and that this was directly related to his personal habits, and that it is a very strong indication for him to stop smoking.

Indeed, the thing that worries me about this gentleman is the rather daunting constellation of risk factors which he shows, and there may be others we don't know about from the given history. I suspect that the only way to deal with this is with a full reform of lifestyle, probably combined with a full assessment, and I feel it would be necessary to ground this officer to get his attention and to obtain his co-operation in what really amounts to a total change in lifestyle, which is not easy, particularly for the typical fighter pilot who is unlikely to be affected by "well intentioned advice."

Dr. Gray: It may be worth sending him off for more detailed pulmonary function in addition to the basic spirometry which has already been done. I'm quite certain that his flow-volume curves will show significant airflow limitation at low lung volumes, and with that information you will have more ammunition when you approach him about stopping smoking. With objective evidence of small airways dysfunction, which you may expect to improve with time after quitting, he may be encouraged to continue as a non-smoker. You could do his small airway function tests now, and then again in six to nine months after stopping smoking, at which time there should be an improvement.

Dr. Giatromanolakis: Do you think the small airways obstruction contributes to the pathogenesis of this x-ray finding.

Dr. Gray: I don't believe the degree of small airways disease he has now had really much to do with the linear shadow, but I'm sure he has some quite independent of that.

Case 3: Arrhythmias
Presented by Dr. Stathogiannis

This patient, a 38 year old engineer, is an applicant for a private flying licence.

His past medical history and family history were entirely unremarkable. Blood pressure, serum glucose, and lipids were normal. He is not a smoker and his life-style is sedentary with moderate daily exercise. He has a history of palpitations since 1966. In 1976, he was submitted to heart catheterization and

coronary angiography in London, for evaluation of the seriousness of his palpitations, with no evidence of coronary or other heart disease.

In 1980, he got a licence for private aircraft (Class B). In 1985, he came back to the HAF Aeromedical Center to renew the flying licence. In this medical examination his blood pressure, chest x-ray and echocardiogram were normal. The resting ECG showed bigeminy and trigeminy, and the exercise treadmill test (12.2.85) was positive for ST changes (2 mm downsloping in D2, 3, AVF, V4, 5, 6). The resting heart rate was 70, and rose to 176 at peak exercise. The resting/peak blood pressures were 120/70 and 215/100 respectively. The PVC's suppressed with exercise, but there were two runs of ventricular tachycardia, of 3 and 4 beats, one and two minutes after peak exercise. He had no precordial pain. The 24 hour Holter showed many unifocal PVC's and sometimes bigeminy and trigeminy, but no runs of ventricular tachycardia.

On September 4th, 1986 he was submitted to stress thallium myocardial perfusion scintigraphy and the results were very good. His exercise tolerance was excellent without angina, ventricular bigeminy disappeared during exercise and the myocardial perfusion during stress was normal.

Topics for discussion:
Flying fitness
Clinical significance
Medication and flight
Suggestions for any other procedures (EPS study, or biopsy)

Colonel Hickman: I hope that he has been returned to his private pilot's licence, because I believe that he would be a very good candidate to grant a waiver to. Prior to 1980, in the US Air Force, we had never granted a waiver for ventricular tachycardia. Our retrospective follow-up of all of our V Tach's who had been grounded (45 at the time) was extremely favourable, for 80% of them. I believe you can imagine the difficulty in going forward to our Surgeon General and his staff, who were largely clinicians, and the only setting in which they had seen ventricular tachycardia was a very malignant one. We were finally able to convince them to start granting waivers, once we were able to demonstrate that we could identify a low risk group.

There were a lot of things that turned out not to be helpful in predicting whether or not they would have subsequent events. The configuration of the v. tach. was not important, the length was not important (although all the runs were very short), the sinus rate at onset did not give us any information, nor did the degree of complexity antecedent to the ventricular tachycardia or on subsequent Holter monitors.

The only thing that indicated any importance at all in the prognosis was whether or not there was coronary disease, valvular disease, or cardiomyopathy.

This young man has had a set of normal coronary angios at age 28, and his is 38 now, and he has a 20 year history of palpitations with nothing more malignant than one three beat run and one four beat run, even though he's got a lot of substrate.

I believe that he does not have organic disease, but my suggestion is that when someone else does the angiograms, you must request to see them yourself, to convince yourself that they are normal, and to make sure that the person who did the angio was sensitive to angiographic prolapse. Although he has a normal echo, that would not deter me from looking at his angiograms again to make sure that I was not dealing with a case of accoustically silent prolapse.

His echo, I am sure, has been reviewed to make sure that he does not have pure apical hypertrophy.

Sometimes if one sees inferolateral ischemia in a young man, one must be suspicious that he might have a circumflex artery anomaly (originating from the right coronary). We see this in about one of every three hundred air force aviators going to cath. If the circumflex passes between the aorta and the pulmonary artery, he could get inferolateral ischemia due to compression. That's why I would always look at the angiograms to make sure that what they thought was a circumflex was not a very small, vestigial vessel, and that the main circumflex did not arise from the main coronary artery, although this is a very remote possibility.

I would like to know about his risk factors so I could assess at age 38 whether I thought that his angiograms at 28 were still good for aeromedical use. He certainly has a normal thallium now.

One of the difficulties of granting waivers for ventricular tachycardia when you have very short runs is that you have no hemodynamic information. Our waivers were limited to seven beats or less because that was the limit of our experience among our study group, but I would rather grant a waiver to someome with a minute of v. tach with stable hemodynamics and no symptoms. In this case, like most of ours, you believe that if he had more he would tolerate it, but you don't know that.

I do not know why he has such an abnormal treadmill, but I do not believe that these are all necessarily what we call "false positives". I think we have a great deal to learn from a metabolic standpoint about why some of these ST segment responses are so abnormal in the face of no big-vessel coronary artery disease, and I think it will take more sophisticated techniques in the future to be able to tell us that.

I personally feel with the data that are available, that he is a reasonable candidate to be granted a waiver for flying.

I don't think he should be medicated, because he has by history and by

repeated treadmill, demonstrated the basic benign nature of his rhythm disturbance, and while we do not totally understand it, I believe that we know that it is almost surely benign. I don't think he needs another cardiac cath at this point unless he has very compelling risk factors I don't know about. I don't think he needs an EPS, or a biopsy.

Dr. Stathogiannis: He never smoked, his lipids were normal, and there was no family history, so his risk factors were low. But, why has he so much ectopy?

Colonel Hickman: I don't think anyone can answer that question at this point. Just since 1968, medicine has newly identified a number of cardiovascular abnormalities including different types of hypertrophy, and mitral valve prolapse. This will probably within our lifetime have a name, and something will be identified, but I think it's unexplained at this point.

Dr. Stathogiannis: Do you expect to see him in a few years with a cardiomyopathy?

Colonel Hickman: I believe that will be one possibility, even a very focal form of cardiomyopathy. I am not sure that there is any information that I could gain from current techniques, like a biopsy or an EPS, that would cause me to treat him differently today, clinically or aeromedically.

It would be nice to know that his ejection fraction rose normally with exercise, and an exercise MUGA would be helpful. We see people with very mild CAD who have slight flattening or a fall of the ejection fraction response at peak exercise. That is why they need to be subjected to a very rigorous upright bicycle exercise, with continuous ejection fractions.

Dr. Gray: Colonel Hickman, are you worried about the ST segment response to exercise? In your study group with exercise induced v. tach. did you have any such positive stress tests, and is this response part of your consideration in granting a waiver?

Colonel Hickman: We did have some with an abnormal ST segment response who did not have coronary artery disease. The ST segment response in the absence of coronary disease had no bearing on the subsequent follow-up which was at least six years.

I agree that we must be cautious about calling this a falsely positive exercise test, because an abnormal ST segment response is distinctly unusual in a population of aviators. Our positivity rate for ST segment response, unselected, is only 7%, and even though a minority of those have coronary disease, it is still an unusual response. I don't think we can just say this is a "false positive." I think we must say that it is an abnormal ST response, and that it is an answer for which we have not yet asked the right question.

Some investigators have shown with flow maps over the myocardium that a significant number of persons with abnormal ST responses but with normal coronary angiograms, also had abnormal flow maps with DSA (digital subration angiography).

One of the things that we have noticed in our lab, is that in persons with exercise ventricular ectopy, most of the VPB's in those who had an abnormal ST response but had normal coronaries came from the left ventricle, just as they did in those who had coronary disease. But in those with a normal ST response with normal coronaries, the VPB's originated from the right ventricle. I believe there is information here, but I just don't think we know what it means yet, which is why I'd be reluctant to call this a "false positive" exercise test.

Regarding follow-up, we would see him every year with a Holter, treadmill and for the first two or three years, radionuclide studies. We would not do a MUGA every year, because that's a high energy isotope. The thallium scan is about equal to a PA chest x-ray, and I would do one every year. After five years, we might stretch out the interval.

Dr. Stathogiannis: Our decision was to grant him his licence, with a requirement that we will see him again next year.

Case 4: WPW Syndrome

A 29 year old fighter pilot (600 hours F-104) was found on routine annual medical examination to have a WPW pattern, type B, on his ECG. He denied any symptoms.

His past history included Gilbert's disease, and a positive Australia antigen, and he had had a liver biopsy. His family history was negative for heart disease.

On physical examination, a short systolic murmur, grade 1/6 was detected along the left parasternal border. This was felt to be functional. Chest xray was normal. The urinalysis and blood chemistry also were normal except for a mildly elevated bilirubin.

Stress test: At peak exercise, when the patient reached a heart rate of 180 bpm, he developed a supraventricular tachycardia (narrow QRS, rate 260 bpm) which persisted for over 30 minutes and was terminated with intravenous verapamil. There was no hemodynamic compromise, and the patient was not symtomatically aware of tachycardia.

The echocardiogram was normal. Holter monitoring was negative except for the WPW pattern.

Topics for discussion:
Flying fitness
Suggestions for other tests
Is this condition a disqualifying and non-waiverable finding?

L/Colonel Kruyer: Since he had no symptoms during the episode of SVT, I would sumbit to him that he does not know that he doesn't do this frequently during exercise or stress, and so a negative history of previous tachycardia is not real helpful. Although there was no evidence of hemodynamic compromise here on the ground, there may well be in a high performance fighter aircraft, pulling G's.

Regardless, we would consider him disqualified from all flying duties because we know he has had at least one episode of sustained tachycardia with a bypass tract.

His ST segment response on the treadmill test is unreliable because of the WPW pattern. If I were to give any possible consideration to a waiver, I would at least want a thallium stress test to look for coronary artery disease. I would also do two or three Holter monitors, and if they were negative, I might repeat his treadmill test one or two more times to look for reproducibility of the rhythm disturbance. An EPS is not indicated because you are not looking at drug efficacy or bypass tract mapping, and there would be no gain in trying to reproduce his tachycardia in the lab.

Question: If he had a negative centrifuge test, would you consider waivering him?

L/Colonel Kruyer: No, I don't think you could waiver him. My suggestion for repeat Holters and treadmills was to try to document repeat episodes as a consideration for anti-arryhthmic therapy, not to recommend a waiver.

Colonel Hickman: I presume the WPW pattern was a new finding, not present when he was applying for flying training. (Affirmative). If you had found the WPW at that time, would you have allowed him to enter flying training?

Dr. Stathogiannis: No, it's easy then. He is not trained.

Colonel Hickman: I am somewhat puzzled; since our rationale for not enrolling a WPW in flying training is because we believe a tachycardia could be quite dangerous, then when we have a tachycardia, why are we not totally willing to remove them from flying? I am still a little puzzled about the decision to not take them into flying training based on an eventuality that they may have SVT, and then when they have SVT, to not permanently ground them. I know that's an arguable decision and if you were going to keep a WPW syndrome flying, you must take them out of high performance. Because you regularly see you aviators in a central location, you have actually discovered this earlier and are safer than we would be.

In the U.S. Air Force, we do not let them into flying training if we find the pattern when they are applying, but once they start training, they do not get another electrocardiogram until age 35, at which time we do discover some with WPW EKG finding. By then, we have 14 years experience with them, and at age 35, we do work them up for arrhythmias and coronary disease if they have WPW pattern. If these are negative, we allow them to fly. We've probably had cases that if we had exercised them in those 14 years between, we might have discovered some WPW syndromes. So, the fact that we in the USAF are in ingornance is not as safe as your seeing them and making the decision to put the ones that you find in non-high performance. I don't want to imply that that is a bad decision, because it is safer than us not knowing because we don't look for it.

We don't put WPW's into flying training for the same reasons you don't, but I'd like to remark on a couple of problems we've had in that regard. We have had applicants for flying training who we have disqualified, and they have gone to a university hospital and had an electrophysiologic study. In one case in particular, they proved, at least as so far as you can prove in the laboratory, that the bypass tract would fatigue antegrade at a very low heart rate, and that it wouldn't conduct retrograde at all. The subject had a vestigial bypass tract, and didn't really have pre-excitation. On the other hand, we do not believe that those who are applying to learn to fly ought to be exposed to invasive procedures. A lot of people don't make it through flying training, and others only fly for five years and then fly civilian, and WPW does not keep them from having a civilian career. So, we have reserved invasive procedures for those who are already trained to fly. If we had 50 WPW EKG findings who applied for flying training, it wouldn't matter whether one of them or 49 of them had pre-excitation, we'd still have to sstudy all 50 of them. We don't feel we can do that in people who haven't yet been trained to fly. Another reason is that once entered into flying training, they require special follow-up, because you are deprived of the use of the exercise test for surveillance of coronary disease, and you must thereafter use radionuclide screening. It takes more medical management than we're interested in.

Dr. Gray: You've already spent a great deal of money training this chap to fly, and he is obviously highly motivated to fly, a real fighter pilot; unhappy about flying

transports. The procedure of sectioning bypass tracts is becoming much more common and succesful, with a low morbidity. I wonder what your opinion would be about his flying status if he had his bypass tract surgically sectioned, with repeated EPS studies and mapping to confirm that the tract was gone, and then with treadmills, Holters and a centrifuge ride, confirming that he could not conduct along the tract.

General Psimenos: This is a very representative case that shows the difference between medical and aeromedical decisions. We took the decision to disqialify this pilot, and this created some tension between the medical community and the pilot community. Pilots think that doctors are eliminating as many pilots as accidents. We had a very hard time explaining even to our leaders what is the problem with this pilot. They would ask questions like "He is not fit for fighter aircraft. He may have some tachycardia duing flight, and a single-seat airplane is not for him, but why do you insist on disqualifying him from co-pilot in a transport airplane? What is the trouble?" Since we do not have any convincing answers, we sent him to Brooks, and you told us of course, that he has to be disqualified from all airplanes, but no clear reason was given for that. What is the actual danger of him dying during flight? Of course, we considered the operation, but what for, to keep him on flying status or to help his life prognosis, because the second indication would be the only one for the operation. But my question is, "Why did we disqualify him from any airplane"?, this special man, with special characteristics.

Lt/Colonel Kruyer: First, Dr. Gray's question about sectioning his bypass tract. If he did that to get back into flying, it would have to be a conclusion that he arrived at by himself and pursued by himself because we would certainly not suggest it, because as Dr. Psimenos said, sectioning is certainly not indicated medically, and I would not do it medically. If he undertook it on his own, I guess we would have to put him back in the air.

As far as putting him back in multi-pace aircraft, in our Air Force the consideration is that during the mission, they all have a job to do... there isn't a pilot and a standby co-pilot to step in if the pilot is incapacitated. They all have something to do, and what they are doing is important to the mission. Another consideration that I think is at least worth thinking about is that if he does develop symptoms during an episode of SVT in the future, even on the flight deck of a transport aircraft he may be a long way from medical attention. If that did happen and he collapsed and got into trouble, the same people that are now saying, "why can't you let him fly a transport airplane" would be saying "why did you let this guy fly transport when you knew this might happen?". I think with all that together, we just wouldn't let him fly.

Colonel Hickman: The three things you have to consider about granting a waiver, are not only whether he will be a danger to other crew members, but whether he'll be a danger to himself, and third, mission completion. I think the difficulty in picking out a favourable case for a waiver, a best case possibility, at least in our Air Force is that we are in a position of basically having to make the same decision over and over and over again. If we did this with our WPW's who had tachycardia, there is no doubt in my mind that we would eventually have a bad event because the numbers would mount up on us. You might get away with it if your case load is very small, but if we put known bypass tracts in multi-place aircraft, we would eventually have an event on the flight deck, especially if we chose WPW's with tachycardia. This problem is far too large numerically for us to take the gamble, because we have so many WPW's.

I think you have to consider that if you did it with this case, you would be placed in the position of having to make the same decision the same way every time, because the aviators expect uniformity and fairness in decision making. I believe that is not totally fair to allow the line to put us into a corner and say "You must totally quantitate the risk for us, and you must totally make us convinced that it will happen, rather than it might happen". The one thing that we know about aviation safety today, and this is not just in people, this is in airframes, avionics, navigational and other aids, is that if you want to make an impact in aviation safety today, you are not going to make a big impact, because we've had 80 years of progress in making sure the odds of events are becoming small. Most aircraft accidents are due to a combination of unlikely and remote events, usually more than one, all coming together at one point in time to produce a disaster. If we're not willing to deal with small probabilities in aerospace medicine today, we won't have any impact.

When I say a confluence of events, I'm talking about SVT at a very, very inconvenient time, perhaps during a very sensitive mission, one that you have only one try at, a mission that you must complete correctly the first time, and you cannot abort the mission.

So, if we're not willing to deal with small probabilities, I don't think we can make an impact in this day and time, because most of the big problems have been very clearly addressed. I think that if our commanders are talking about aircraft parts and airframes, they will accept almost no compromise, no probability of failure. I don't think that we can be held responsible for grounding people who ultimately do not die, or do not develop the complication that we grounded them for.

Dr. Gray: In our Air Force, as I think in most, the medical branch are advisors to the operational side, and although we sign the papers and say "they're grounded", that is really a recommendation, and what we are saying is "Here is our aeromedical

assessment of the problem and the risks involved, to your mission, and to the individual" It is still the final decision of the operational side to assess that risk and decide whether or not they require that individual to fly.

General Psimenos: In the Greek Air Force, the decision of the Aeromedical Center and the Supreme Medical Committee is final and compulsory for the operational people. It cannot be bypassed, even by the Minister of Defence. So, we have this authority, and of course we always try to come to the right conclusion, and not give them any right to doubt about the specificity of our decisions. But this case was a real challenge, because the possibility of something happening is very remote, and we have a young pilot now in the combat wing who is complaining that the doctors were very strict with him.

Colonel Hickman: I believe we must do everything possible to discourage the performance of dangerous procedures and interventions for purely occupational reasons when they are not medically indicated. If people start to do this, we will get increasing numbers of people having dangerous things done to them before they learn to fly, only to find they are disqualified for some other reason, or, that they don't have the ability to learn to fly. We are going to see some very sad things start to happen as more and more people have things done to themselves so they can fly. I think we must really try to discourage that.

Case 5: Atrial Fibrillation
Presented by Dr. Masdrakis

Colonel PJ is a 45 year old aviator of the Hellenic Air Force who was admitted to hospital on February 14th, 1986 because of atrial fibrillation that was found during the annual periodic examination.

The patient was asymptomatic and was not able to say when the arrhythmia started. There was no history of diabetes, hypertension. No family history of heart disease.

He is a non-smoker, but he was under stress because of his job. On examination, his weight was normal. Auscultation did not reveal any murmur. Chest x-ray showed a normal heart size. The ECG showed atrial fibrillation.

An echocardiogram showed normal valves, mild dialatation of the left atrium., and normal ventricular function. Laboratory findings were normal, including lipids (cholesterol 149 mg/dl, triglycerides 80 mg/dl) and thyroid function.

Therapy was started with propranolol, anticoagulants, digitalis and quindine, but with no effect. On February 24, cardioversion was performed with 50,100 and 150 joules, but was of no effect. Therapy with digitalis and quindine was continued and on March 3rd, cardioversion with 300 joules was effective in restoring sinus rhythm. The patient continued to receive oral medication for one month.

The exercise test was normal. There was no ST depression, and no arrhythmias.

After two months, this officer returned to flying status without medication. We did not perform electrophysiologic studies or angiography, because we felt his low risk factors and negative exercise test made it unlikely that he had coronary disease.

Topics for discussion:

Flying Fitness
Medication and flight
Suggestions for any other test and procedures

L/Colonel Kruyer: He had no hemodynamic symptoms, so we cannot be certain how long he was in atrial fibrillation. Since he is 45 years old, our evaluation would have included coronary angiography before considering a waiver, because of the concern that even minimal coronary disease might precipitate ischemia with the occurence of the tachycardia in a stressful situation. This would be required in any aviator over the age of 35. If you were going to stop short of angiography, I would at least want a thallium study, but in our experience, over the age of 35, even a normal stress test and thallium was not reliable in excluding coronary disease.

We would ground him for six months observation, looking for clinical recurrence and obtain during that time three Holter monitors, before doing a catheterization for purely aeromedical reasons. If he had any degree of coronary artery disease, he would be grounded.

If his coronaries were clean, we would proceed to an electrophysiologic study, to see if we could induce the arrhythmia. If, we could, with single, paced ectopics, we would assume he is at high risk for recurrence, and he would be grounded. We have only been able to induce tachycardia in individuals like him, who had a prolonged, sustained episode, usually of atrial fibrillation. So, we still do EPS studies in people like him, primarily to see if we can reproduce the rhythm disturbance.

His ventricular response during atrial fibrillation was relatively slow. In clinical cardiology, this is sometimes a sign of AV nodal disease, but in otherwise healthy aviators with atrial fibrillation, it is not uncommon for them to have a slow ventricular response, probably because of a high degree of vagal tone in a healthy AV node. This can be demonstrated by exercising them.

In this case, he had a long period of atrial fibrillation without symptoms of hemodynamic compromise. In some ways, this is more comforting than in people who have short runs, in whom we can't be sure about in the hemodynamic consequences of more prolonged episodes.

I would not recommend any medication at this point. You rightly left him on medication for a few weeks, to ensure that he stayed in sinus rhythm, and since he had only the one documented episode, there is no indication for chronic suppressive therapy. In the absence of underlying disease, with no recurrence, I would not treat him.

Dr. Masdrakis: Suppose that the patient has a 20 to 30% obstruction in one or two coronary arteries. Do you think that, for transport airplanes, he must be disqualified?

L/Colonel Kruyer: With a 30% lesion by itself, we could waiver him, or, with the SVT by itself, if the remainder of the evaluation was normal (including coronary angiography), he would be fully waiverable. With the combination, we would still require that he be grounded from any flying, including transports and bombers. Grading of the severity of lesions is highly variable, and the anatomic severity does not always correlate with the hemodynamic severity, do that even a 30% lesion might become hemodynamically significant, when combined with a tachyarrhythmia, especially in a stressful situation, which is difficult to test.

Colonel Hickman: The issue of coronary disease in SVT eliminates only 15 to 20% of our SVT study group aviators, so it does not eliminate a great number, but very quickly eliminates those we are concerned about.

If you grant a waiver for SVT, I believe you have to go on the assumption that it will happen again. Therefore, I must know conclusively whether or not he has coronary disease, which bears upon the sensitivity of thalliums. The way we do thalliums at Brooks is the day before they have a thallium, they have a maximal symptom-limited exercise test, so that we can see exactly how long they are going to exercise. When we do the thallium the next day, we can inject the isotope exactly one minute before they went to exhaustion the day before, to increase the sensitivity of the thallium test. This also allows us to get recovery tracings, which we can't immediately after the thallium. Even under those circumstances, we do not trust the thallium to totally exclude coronary disease in a situation where we must conclusively know whether it is present or absent, even in minimal degree. In this setting, the false negative rate of thallium scintigraphy is too high.

Since we have started doing fluroscopy on our aviators, we have found that if we have positive coronary fluroscopy with calcification of coronary arteries, but a negative thallium and a negative treadmill, 34% of those aviators have had a 50% or greater lesion. We have 40 or 50 aviators in this group, and this is starting to give us some concern about the false negative rate, and we feel we just have to go ahead and do the angios.

Case 6 - Coronary Artery Disease and Hypertension

This is a case of a civilian airline pilot, 57 years old. In 1981 he was first noted to be hypertensive, but his blood pressure gradually climbed over a 4 year period to 180/100 despite treatment with diuretics.

An exercise test performed in September 1985 was strongly positive and for this reason he underwent a coronary angiogram in London in October which showed the following:

a. Normal sized left ventricle. Good ejection fraction. No mitral regurgitation and no mitral leaflet prolapse. No calcification. Normal left ventricular pressures.
b. Left main anterior descending and circumflex coronary arteries showed subcritical narrowings with a 40% stenosis at the origin of the first diagnonal branch.
c. Right coronary artery normal. Dominant vessel. No cross filling.

In the past six months the patient has clearly been complaining of angina pectoris provoked by strenuous exertion, although he does have flight insurance and we are not sure about the validity of his symptoms. A second exercise test performed in July 1986 was stongly positive too. At the same time he underwent a thallium scan which was normal.

His hypertension was controlled with atenolol 50 mg and nifedipine slow-release 20 mg twice daily.

Topics for discussion:

Flight and hypertension
Medication and flight
Flying fitness
Flight and coronary artery disease

Colonel Hickman: As you know, we have been returning aviators with minimal coronary artery disease back to non-high performance flying for about 10 years. They have to be totally asymptomatic, and we don't accept any disease in the left main at all. Even as the angiograms are reported from London, that would have exceeded our threshold for minimal disease. He also has angina, but you have a problem here that you have already alluded to - the evaluation of airline pilots who at this age are well insured. This sometimes raises the difficulty of proving not that they are unfit to fly, but proving to them that they are fit to fly.

It has been 18 months since he had angios, and after reviewing the cines, I believe the diagonal lesion is worse than 40%, and I believe there is also a lesion that exceeds 70% in the posterolateral branch. I believe the only answer at this point is to recath him, and to do all the views that you want. I believe that you are going to find a degree of coronary disease significant enough to explain ischemia. Whether or not he has angina is difficult to say.

If one wanted to be absolutely scientific, one could put a catheter in the coronary sinus, and pace him to see if he produces lactate, but I wouldn't do that. I would just repeat the angios, because whether he has angina or not, if he has lesions that exceed 50% in his diagonal and a branch of his right coronary, to me that would be information enough.

I think if you have done the angios on this aviator, he would have had more views a a more complete study. We have the same problem with angios done on National Guard aviators, and although we don't insist that all angios be done at Brooks, we do like to talk to the cardiologists who do catherizations for occupational reasons to make sure they are aware of the implications, and that they are attuned to minimal disease and do the necessary views.

Question: The coronary disease aside, do you think his hypertension is waiverable, controlled with atenolol and nifedipine?

Colonel Hickman: I'm sorry, but I don't feel adequate to answer that, since my total experience in treating aviators with hypertension has not been expanded to treating them with calcium blockers and beta-blockers. I would be reluctant to have an airline pilot on nifedipine, and I think it would take a considerable period of observation to make sure that the vasodilatory response was one that was highly predictable.

L/Colonel Kruyer: I think the previous angiograms show a significant diagonal lesion, and if I was convinced that he had angina, I would not repeat the cath unless I was going to pursue angioplasty or by-pass surgery. His symptoms could probably be controlled with medication, and there is no definite indication for either angioplasty or bypass. Should he consider either, I would have a very careful discussion with him about the risks, because I have personally seen two patients die on the table while having a diagonal lesion angioplastied.

Case Discussions - Denmark

Case Number 1
Presented by Dr. Sandoe
(This case was not recorded on tape)

57 year-old airline Captain (commercial) born 1930. No cardiovascular complaints. Never any sensation of palpitations. Functional capacity good. Runs 10 miles five to six times a week.

Primary abnormal finding

At the routine examination with exercise in 1985, ventricular ectopics from two to three foci (episodically as bigeminy)

Supplementary investigations:

Clinical investigation normal. BP 130/80 mmHg.
24-hour ECG monitoring (portable tape recorder): CA 300 ventricular ectopics/24 hour from three foci. One short episode (four complexes) of accelerated idioventricular rhythm. One couplet (monomorphic)

Chest x-ray: Normal heart size, normal vascularity of lung fields.

Echocardiography: Left ventricle slightly dilated (diastolic diameter 6.2 cm, systolic diameter 4.2 cm), normal FS (32%)

MUGA: Ejection fraction at rest normal (43%) but no increase in EF during exercise (with a) stepwise increased workload up to 150 watts for 5 min, with increase in heart rate up to 160/min and unchanged EF (45%). Both at rest and during exercise, hypokinesia of basal and lateral part of the left ventricular wall.

Thallium scintigraphy: No myocardial perfusion defects at exercise (150 watts for 4 min, increase in heart rate up to 140/min).

L/Colonel Kruyer: This 57 year old man has ventricular ectopy and evidence on the echocardiogram and MUGA of a possible myopathy. While we consider ectopy alone on a routine treadmill to be acceptable if there is no evidence of underlying heart disease, this is not the case here.
Both a cardiomyopathy and coronary artery disease are possible concerns here. He has developed ventricular ectopy and evidence of left ventricular dysfunction, and aeromedically, we would ground him based on the available information. I would recommend that he undergo cardiac catherization for purely clinical reasons, to clarify the diagnosis and prognosis.

Dr. Sandoe: We would also disqualify him from flying here for the combination of progressive findings. We would not do a catherization because clinically we would not treat him, and the results would not alter our aeromedical disposition.

Case No. 2

(This case was not recorded on tape)

44 year old traffic controller born 1943
No cardiovascular complaints
Functional capacity normal. No sports activities during leisure time.

Primary abnormal finding:
Routine exercise ECG with horizontal ST depression in Leads II, III, V3 to V6 (up to 2 mm starting at a heart rate of 140/min).

Supplementary investigations:
Clinicical investigation normal. BP 140/80. Resting ECG normal

Echocardiography:
Normal. No ventricular hypertrophy or dilatation. FS normal (38%).

MUGA: EF at rest 56% with a decrease to 47% during exercise (125 watt, 9 min, max heart rate 162/min and max BP 270 systolic)

Thallium scintigraphy: No perfusion defects (stepwise increase of exercise for 14 min up to 200 watts: max heart rate 174 and max BP 250 systolic).

Dr. Sandoe: Was it a mistake to do the exercise test?

Colonel Hickman: We really can't make the decision about whether it was a mistake to do the exercise test without a knowledge of his risk factors.

We have two abnormal tests that suggest coronary artery disease, the exercise ECG and the exercise MUGA. However, we need to know more about this man's risk factors for CAD. We have not been impressed with the ability of the MUGA to detect coronary artery disease, and with the normal thallium, if his risk factors are low, the exercise ECG may well have been a false positive. If he is at risk, it may well be true positive, especially if the changes in the exercise ECG have been serial. At this point with this information, he will need coronary angiography.

Dr. Sandoe: In Denmark, we apply a more general application of the exercise test, beginning at age 30. We find about 5% abnormal, and they then have an echo, Holter, thallium and MUGA.

Colonel Hickman: The sensitivity and specificity of the thallium and MUGA are known, and the predictive value of both tests depends on the prevelance of the disease in your population. We have found that the thallium is not as good as we thought in our original paper in 1980, and the predictive value will suffer the same fate as that of the exercise ECG in a population with low disease prevalence. In Denmark, your routine screening exercise ECG prompts a thallium and MUGA in 5% of your population. We would prefer to save the nuclear studies for a more select population.

Dr. Alnaes: In Norway, our approach is also to do regular screening exercise ECG's at three yearly intervals after 40. From 1983 to 1987 we had 40 with ST depression or pathologic Q waves, who underwent a MUGA, thallium and risk stratification. 22 were at low risk and still fly, and 18 were at high risk and were disqualified from flying. We do not do coronary angiography in asymptomatic people in Norway. Six were subsequently cathed with symptoms and underwent coronary artery bypass grafting.

Case History 3

56 year old airline captain (commercial) born 1929

No cardiovascular complaints. Runs 5 to 10 miles two to three times weekly. For several years intermittent complaints of pain in the right knee during physical exercise, in particular hiking, which makes it impossible/difficult for him to work for more than a few minutes on the cycle ergometer.

First episode with abnormal findings:

Normal exercise ECG at a routine test in 1976. However, at the next routine test in 1979 at a work load of 150 watts (max heart rate 160) he developed an attack of monomorphic ventricular tachycardia (rate 210/min lasting for 36 seconds). In the weeks up to the test, he had fever and catarrhalia. He was grounded for four months, but allowed to fly again after:

- preliminary lab tests (CK MB, LDB, Hgb, sedimentation rate, AST, ASH) were found normal
- normal echocardiography and chest x-ray
- electrophysiologic studies showed normal atrioventricular and intraventricular conduction, and re-entry tachycardias could not be induced by pacing
- and last but not least, a new exercise ECG (work load stopped at a heart rate of 120/min due to pain in right knee) which induced no ventricular ectopy.

Second episode with abnormal findings:

Following a normal exercise ECG in 1982 and 1984, a new exercise ECG in 1985 induced an 18 second attack of monomorphic ventricular tachycardia of the same QRS configuration as in 1979, but now with a heart rate of only 130 to 140/min.

Supplementary investigations:

Clinical investigation normal. BP 130/80

24 hour ECG monitoring (portable tape recorder): 500 to 1000 monomorphic ventricular ectopics/24 hours. Sometimes period of bigeminy, no couplets.

A new exercise ECG 1 month later up to a heart rate of 120/min and BP of 220 systolic did not induce tachycardia, but showed episodes of monomorphic ventricular extrasystoles periodically as bigeminy (same QRS configuration as in the former attacks of VT)

Echocardiography: Normal findings

MUGA: Normal (resting EF 47% at a heart rate of 59/min, following exercise a rise in heart rate up to 118/min, in BP up to 160 systolic and in EF up to 63%)

Thallium scintigraphy:

Normal, without perfusion defects (exercise up to a heart rate of 160 and a systolic BP of 210)

Question: Does he or did he ever have any symptoms of tachycardia?

Dr. Sandoe: No, he had no symptoms during the attacks, and feels perfectly healthy otherwise.

L/Colonel Kruyer: According to the rules in our Air Force, he would have been grounded following his initial episode.

Dr. Sandoe: When I first saw this ECG, I agreed that he should not be flying. But couldn't he be a co-pilot?

L/Colonel Kruyer: That depends on your concept whether or not the copilot is performing a critical crew function, or is a redundant crewmember ready to take over if the pilot is incapacitated. The other problem is that if he has a cardiac arrest during flight, you still have to get him back down to the ground.

When he initially presented the thought may have been that he had a viral myocarditis and had V Tach on that basis. From our experience, the episode was too long. He didn't just have one episode of V Tach, he had 36 seconds of sustained ventricular tachycardia and a repeat episode of V Tach. I wouldn't want to fly with him.

The initial assumption was that he had myocarditis and not coronary artery disease, but nothing was done to definitely rule that out. We now have eight years of follow-up so CAD is less likely. Coronary angiography is generally done to ensure normal coronary arteries before electrophysiologic studies are done to try to induce ventricular tachycardia. I likely wouldn't have done the EPS study, because I would have disqualified him from flying. If on his initial episode he had 7 beats or less, we would have done coronary angiography and if that was normal we would have returned him to flying duties. Repeat episodes on further treadmills, as long as there were under 7 beats, would not have been a problem and he would have been able to continue flying. some clinicians would only define sustained ventricular tachycardia if they have had to use cardioversion.

Colonel Nickman: One of the things that I wanted to add was about feeling reassured if he were to fly as a co-pilot. I am sure one of the reasons that he was allowed back flying was because there was another pilot in the cockpit. I believe that in terms of aircraft accident prevention that the concept of crew redundancy has been over-sold. The overwhelming majority of aircraft accidents occur during transition phases of flight, either during take off or landing, when the aircraft is closest to the ground, and when Holter monitoring of commercial pilots has shown their heart rates to be the highest, and so during which this pilot would be most vulnerable. It is the period of time in which the margin for the other person to react is the smallest because they are in the transition phase of the flight. Flying straight and level at 37000 feet is okay, but almost no accidents happen there unless you run into another airplane. Most accidents happen during phases of flight in which redundancy of the crew is much more questionable. I know that they get emergency training, but they are expecting an emergency in the simulator at some point, and it is a contingency that they can plan for.

Basically this pilot was allowed to continue flying because the person responsible felt that this was a normal variant. Now 8 years later, you can say that with somewhat more assurance than after 4 months of observation. But I think to say something is a normal variant in a clinical population is okay, and you don't really have to prove to the Nth degree that it is, but for an airline captain in order to say that ventricular tachycardia is a normal variant requires a lot more workup. A left ventriculogram and coronary angiography would have answered most of my questions about whether or not he had diagnosable underlying heart disease. In someone with exercise induced ventricular tachycardia that I want to return to flying, I would not trust the echocardiogram or my ears in this situation to make absolutely sure that I was not dealing with mitral valve prolapse. However, coronary disease would be the main t .g that I would want to know about.

.is electrophysiological study in my opinion does not rule out in any way the possibility that early in the game he had organic heart disease. If from the normal variant stand point, you don't expect the captain's health to be any better than those in the back of the airplane, there is not much use in having physical standards. The "man-off-the-street" argument which says that "normal" people have this is not acceptable. I want better health in my aircraft captain than in the "man-off-the-street". If I was going to accept a "man-off-the-street", I wouldn't need any physical standards.

If this captain were in an aircraft accident, and the cause of the accident could not be conclusively decided, how would your aeromedical authorities explain this away? I think that even the public can understand this kind of case. I think that if they got the facts on a case like this, after an aircraft accident, I think it would be a terribly difficult thing to explain.

Question: We have discussed the high performance pilot, and the commercial pilot but what about the private pilot with a condition like this, would you consider him fit for flying?

Dr. Sandoe: I wouldn't be in any doubt, I would not consider him fit to fly. The only excuse for him to fly is that he is in a cockpit where others can take over, but then you come back to the problem that has never been solved, that is, should the rules be stricter or less strict for private pilots flying in a single cockpit.

Colonel Hickman: Well, a private pilot shares the airspace with the rest of us, and I don't have to be right in the cockpit with him to be at some disadvantage in controlled airspace. The last two major accidents in the US involved airliners and private planes. I do not feel better because the only life they feel they are risking is their own. I wouldn't let the private pilot off the hook on that basis.

Question: What is that status of this pilot today?

Dr. Bonde-Peterson: He is flying with another pilot, controlled with regular Holter monitors.

Dr. Sandoe: Yes, but a Holter monitor is only significant if it is positive. A negative finding doesn't really help.

Colonel Hickman: The only way that we were able to get aviators with short runs of ventricular tachycardia back to non-high performance flying was to show in a prospective follow-up study what the discriminators were. One of the discriminators that was of no help were reproducibility of the rhythm disturbance. Anything that you could look at from Holter and stress tests were of no help. The only thing that was of any help was to identify whether or not there was structural heart disease. This has been answered non-invasively in this case but hasn't been answered fully enough to suit me. The only way that we would let V. Tach back would be with a coronary arteriogram.

We would probably have picked this individual up in our system. If someone has a VPB that you capture on an annual EKG and you start figuring out how long it takes to do an EKG, what is the probability that he has infrequent ventricular ectopy? Well the probability that it is infrequent is very small. The Israelis did a study and showed that if you have a single VPB on an annual electrocerdiogram, and you work that patient up, then nearly one-third of them will have complexity.

Dr. Sandoe: Yes, but this is the point, he did not have any VPB's on his resting electrocardiogram, and his problem was only detected because of the routine exercise test.

Case No. 4

40 year old co-pilot (commercial) born 1947
No cardiovascular complaints
Functional capacity good. Jogs for half an hour three to four times a week.
Never experienced attacks of palpatations before. No syncope.

Primary abnormal finding:

Normal routine exercise ECG in 1977, but at a new routine examination in 1987 an attack of supraventricular tachycardia lasting for 23 min after stop of work (start of tachycardia after 5 to 6 min of work up to 200 watt, heart rate during supraventricular tachycardia 180 to 210 with sudden acceleration to 250). Sudden stop of tachycardia, change to a heart rate of 90/min at 23 to 24 min after the stop of work. Tachycardia was most likely of AV junction type.

Supplementary Investigations:

Clinical investigation normal. BP 140/90
Hgb, sedimentation rate, CK MB, LDH normal

Repeat exercise-ECG two weeks later: Stepwise increase in exercise load up to 200 watt over 6 min. Maximum heart rate 165. No attacks of tachycardia. No signs of ischemia

Chest x-ray: Normal

Echiocardiography: Normal

24-hour ECG monitoring:

(portable tape recorder). A few supraventricular ectopics. No episodes of tachycardia.

MUGA and thallium scintigraphy: Normal

Follow-up 3 and 6 months later:
ECG normal. No history of tachycardia. ECG normal
24-hour monitoring: A few supraventricular ectopics

Dr. Gray: It is reassuring that he had no symptoms during the tachycardia, that is no presyncopal symptoms, so he was able to maintain an adequate cardiac output even with the very high ventricular response which at times was as high as 240. He appears to be a very fit fellow with a high estimated oxygen uptake, so he otherwise appears to be a pretty healthy guy.

There are really two concerns here. One is whether he is going to get this again, that is what is his risk of recurrence, particularly at a critical time in the air which could be a problem as far as flight safety goes. Thinking about the risk of getting it again, the one question that I have is whether or not he might have a concealed bypass tract, which would be a concern especially since his ventricular response during SVT apparently changed rather rapidly from 180 to 250. For some reason, his ventricular response sped up during SVT, so I have some concern that he might have a concealed bypass tract which was intermittently conducting. However, there is no other evidence of it except for the rapid change in rhythym.

The other concern is that he might have underlying coronary disease which could be exacerbated by SVT. It is reassuring that he has a normal thallium study, and MUGA scan, and those two non-invasive studies both help to reassure us that he doesn't have CAD, but on the other hand, he did have ST changes during the tachycardia, and immediately afterwards. In the 2 minutes post exercise tracing, he has at least a millimeter of ST depression. So I still have some concern about the possibility of underlying coronary artery disease.

The questions that I have, then, relate to the type of flying that he is doing. He is a commercial co-pilot, and the additional investigations that you might want for him are a little different than you might want for somebody that is flying a high performance fighter.

The questions are does he need to have an electrophysiologic study, and secondly does he need to have coronary angiograms done? I think that if he were flying one of your F16's, at age 40, with the information that you have here, before you put him back into the cockpit you would want to do an electrophysiologic study to see if he has a concealed bypass tract and to evaluate how readily his SVT might be reproduced, and even though he had a normal thallium study, since he had ST depression during the tachycardia, you would want to do a coronary angiogram. You would probably also want to take him down to Soesterburg and spin him in the centrifuge to see if he got SVT under increased "G" forces. That would be the kind of investigation that I would want to see done if he were a F16 pilot.

Since he is a commercial co-pilot, I think that the investigation would be a little different. With the information that we have here, given that his thallium study is normal, and he hasn't had any recurrence in six months, with follow-up including additional stress tests and Holters, probably in our system at least, he could go back to flying in an "as or with copilot" in the commercial world without any additional studies being required.

Dr. Sandoe: Are there any comments from the USAF?

L/Colonel Kruyer: If he was a high performance flyer, before he could be reconsidered for flying duties, he would have to have a monitored ride on the centrifuge before he had any invasive test. If he had repeat SVT on his Holter or stress tests, or on the centrifuge, he would be grounded from military flying. Being over age 35, even with normal non-invasive studies, he would have coronary angiography. If his coronaries were normal, he would have an electrophysiologic study.

Question: Since most aircraft accidents occur during the transisition phase, what is the meaning of an "as or with copilot" restriction, since it is unlikely that the other pilot will have time to take over?

Colonel Hickman: I think that we are basically treating ourselves with such a restriction. I think we are rearranging the deck chairs on the Titanic. It makes us feel better, but we are really not helping. I think that it is cosmetic in nature.

Dr. Sandoe: I think that in this case that the copilot restriction is a good idea, because this individual showed that he can take 18 minutes of supraventricular tachycardia, which he tolerated well. If you have ventricular tachycardia, you always have the risk that it could convert into ventricular fibrillation, but we could say that this man most likely could keep on for at least a few minutes until there would be time to take over.

Colonel Hickman: If he had normal coronary angiography and a normal EPS, I would put him back flying into any aircraft. I wouldn't hedge my bets any by saying let's go copilot. I think when you have a case of SVT like this you have to assume that there may be a recurrence, and although he tolerates it well today the disease that a 40 year old male is most likely to have is coronary atherosclerosis. With SVT, even mild low grade lesions may grow, and the biggest disease he may not tolerate with SVT is coronary artery disease. I don 't think the thallium is sensitive enough to pick up small degrees of coronary artery disease. We don't put anybody back flying unless they have pristine coronary arteries. They can't go into minimal coronary artery disease protocol, because we know the disease that they can least tolerate in the long haul is coronary disease. I don't think that thallium detects disease at a low enough level that I would be satisfied with it. I would have to know that he had pristine, normal coronaries and then I would put him even in the F16.

Dr. Alnaes: But if you can't have the answer.

Colonel Hickman: Then I think that you have done the best that you can under the circumstances.

Dr. Almaes: Taking up the question of copilot, we had an infarct in a Hercules approach to Keflavik in Iceland in November in a winter storm. It was about as bad as it could be, and the plane was full of ammunition. There was an infarct on final approach, about a mile out from landing. The copilot took over and landed the plane. He even had time on the final approach to call up the ambulance so it was standing by shortly after they landed. The plane could not have been saved unless th re was a copilot there to take over. So there is this redundancy and it should be ained for. It is not completely academic.

Dr. Sandoe: During the lectures you discussed the question of "holiday heart" and indicated that after a period of 6 months, you would return such a pilot to flying without the requirement for coronary angiography or EPS. What is the difference between that situation and this pilot?

Dr. Gray: The difference is that during his tachycardia, this pilot demonstrated ST depression which raises the concern about coronary disease.

Case No. 5
44 year old airline captain (commercial) born 1941
Formerly healthy. Never previously hospitalized.
Never before experienced pain in the chest.

November 19, 1985: Fever (39.5 Clecius), tonsillitis, start of treatment by GP

November 21, 1985: Sudden attack of chest pain located retrosternally, radiating to both arms. Admitted to hospital. ECG showed initially ST elevation in leads I, II and V4-6 with a gradual lowering of the ST segment towards the isoelectric line combined with T wave inversion over the subsequent days. No development of Q waves. After 2 weeks discharged with a diagnosis of pericarditis, but with a remark that there had been a significant elevation of LDH (fraction I, the heart component of the LDH complex), which might support a suspicion of acute myocardial infarction. Over the following months intermittintly but repeatedly complains of retrosternal oppression not related to exercise or stress.

Supplementary investigation
(3 months after the acute illness):
Exercise ECG: High exercise capacity, no ischemic changes, no arrhythmias (load up to 200 watts over a period of 12 min, maximum heart rate 170/min and maximum BP 200 systolic).

Thallium scintigraphy:
No perfusion defect (exercise load 200 watts for 3 min, heart rat 170/min).

Chest x-ray: Normal

Echocardiography:Normal

MUGA: Normal (EF at rest 51%, during exercise 64% - 150 watts, maximum heart rate 160/min)

Dr. Gray: The diagnosis is almost certainly pericarditis based on the information available. However, there is some concern that he has coronary disease. But, the major concern is that he still has symptoms now some months after, worrisome not only because they might represent coronary disease, but worrisome in relationship to his duties as an aircraft Captain. Whether or not it is related to coronary disease or persistent pericarditis, if he is on final approach into Kennedy, and the weather is bad, and he has this oppressive chest pain that is bothering him, I certainly wouldn't like to be sitting in the back of his airplane. So, the fact that he is still symptomatic is a worry, even though the initial diagnosis is pericarditis. The observation that his LDH1 was up raises a couple of possibilities. He might have a bit of myocarditis, or another is that it might have been a lab error. I assume that no other enzymes were up and, given the information, I would assume that he had a mild myocarditis.

The question as to whether or not he has ischemic heart disease and whether this might have been a subendocardial MI still looms. The non-invasive studies that you have done certainly helped to rule this out, but again, they are not 100 percent sensitive. Based on the non-invasive studies, it seems unlikley that he has coronary disease. The factors that I would consider in this case are firstly that he is civilian rather than military, and again the workup and disposition are somewhat different, even though from the purely aeromedical point of view it should be similiar, but civilian authorities often have a differnt approach than military. I know that in Canada at least, it would be unlikely that he would have to have coronary angiography. However the fact that he is still symptomatic is most bothersome, and I think that he would be required to stay on the ground until his symptoms have gone away.

If he were a military pilot, I think that we would want to do coronary angiography to prove to us, and to him, that he does not have coronary disease.

So I think that would be the disposition in Canada. We would want to ground him until his symptoms went away, and depending on whether he is military or civilian he might be required to have angios.

Question: What would you do in the USAF?

L/Colonel Kruyer: I would want to take a very careful history myself. If I though there was any question of this atypical chest pain being angina, I would have to cath him. Even as a patient in a non-aeromedical sense, if I thought he might be having angina, I would cath him. If I was convinced that this was non-ischemic pain, but was pericardial pain, I wouldn't cath him. But I wouldn't let him fly in the military either, because he still has symptoms.

Dr. Sandoe: What about the RAF?

G/C Hull: I would be unhappy about him flying because although one could say the pain was just pericardial, I don't know how long you could say that it wasn't pericarditis that was going to deteriorate suddenly. So I think I would want to watch him whilst he was still symptomatic. And I think that if there were any doubt at all, one would want to do angiography.

Case No. 6

27 year old applicant for commercial airline certificate, born 1959. No cardiovascular complaints. Never any attacks of palpitations or syncope.

Primary abnormal finding:
Routine ECG showed WPW pattern (concertina effect). Repeat ECG showed normal findings.

Supplementary investigations:
24 hour ECG monitoring (portable tape recorder): WPW pattern alternating with a normal QRS pattern with a PR of 0.14 and no delta wave.

Echocardiography and Muga: Normal

Electrophysiologic investigation:
Accessory AV conduction septally placed near the AV node. Conduction through the accessory AV connection only retrogradely. It was not possible to induce re-entry tachycardia by pacing. Normal antegrade AV conduction. Normal intraventricular conduction.

Exercise ECG: High exercise capacity (maximum heart rate 188, maximum blood pressure 225/100. No WPW pattern during exercise. No attacks of tachycardia).

L/Colonel Kruyer: I am not sure what would happen with the commercial pilot applicant, but I will comment on the USAF approach. As an applicant with WPW, we would not train him to fly. He has an EPS study which shows that he does not conduct antegrade down his bypass tract so that presumably, if he goes into atrial fibrillation, he will not have a real rapid ventricular response. However, at some point at least, he did conduct antegradely down his bypass tract or he would not have had a delta wave, and he would have never been diagnosed. So the EPS does not help you a whole lot. He does conduct retrograde, so that he could have a re-entry tachycardia. They could not provoke it in the lab, but that was under resting conditions, maybe sedated, and supine in the cath lab, and it (the propensity for re-entrant arrhythmias) is not a constant steady state thing. It is an interaction between the conduction velocity, the refractoriness of the AV node right now, and the bypass tract right now and the relationship between the two under whatever sympathetic and parasympathetic tone that is going on, and when the PAC occurs. To induce a bunch of PAC's in the lab and say that we couldn't induce SVT and so this guy will never have SVT with WPW, I just don't think that you can say that.

Dr. Sandoe: So you wouldn't let him train. No one would let him train?

Colonel Hickman: If you could show that he had pre-excitation at heart rates that were only modest, that would be a helpful piece of information. If you could show that he wouldn't conduct retrogradely at all, and that he would fatigue antegrade over and over again at heart rates of 80, 90 or 100, at that momemt that you tested him, he would not have true electrical pre-excitation. It would be a much stronger argument than is the case here.

Dr. Sandoe: Would you then take him to train?

Colonel Hickman: Very reluctantly, but I guess we would have to. The thing that bothers me about cases like this is that they go out and get this test. It is an easy enough problem if you only have an occasional WPW that wants to fly. But from the USAF Academy alone, we have lots of guys who come into their first year in the academy and they show WPW, and they know from that momemt they can't fly. But there will be a half a dozen or so that will have a pattern that emerges between their first year and last year. If we started choosing people with WPW for pilot training based on electrophysiologic studies, we would be faced with the prospect of doing 30 to 40 EPS studies yearly in young men who have not learned to fly and who only want the opportunity to learn a dangerous occupation. Now for most university hospitals, that is an interesting case that they do one of and it sounds good. But if you had to do this 40 times a year, in healthy young men just for the opportunity of learning to fly, in our situation, one out of 4 would prove not to have the requisite skills to learn to fly, and the big majority would leave the Air Force in 5 years. We are just simply not going to be involved in doing procedures of risk in young ment just for the opportunity to learn to fly. We reserve invasive procedures for those who are already trained to fly.

I really have a lot of heartburn about having young men who want to learn to fly in this sort of situation. We were faced with a situation similiar to this and we were more or less pushed into accepting him for pilot training. He had an electrophysiologic study done at a university hospital. But if the guys at the university were faced with doing this year in and year out, they would soon lose their enthusiasm for it. But they haven't understood that this guy hasn't learned to fly an airplane yet. He may be a total klutz and not even know how to drive a car. But they don't know that. And we are not turning pilots out for the American airlines. We are turning them out for the airforce.

Dr. Kruyer and I had a case forwarded to us from another country in which they showed us an electrophysiologic study that was very compelling and they and they concluded from this that he did not have significant pre-excitation. In the same package was a treadmill that showed him a wide QRS tachcardia. The electrophysiologist concluded from the EPS study that his WPW was benign and in the same package, the guy that worked him up did a treadmill, and there he was, in a WPW tachycardia. We have come to sort of distrust the EPS study in that regard.

L/Colonel Kruyer: Plus, tnen these candidates may, after they have had their EPS study, go find someone to ablate their bypass tract so that they can get into pilot training, and that is really going way too far.

Dr. Sandoe: Would you take a man who had been operated on for his bypass tract?

Colonel Hickman: Absolutely not. I think that shows judgement too poor to be giving him a 25 million dollar airplane, to have your heart operated on to learn to fly. That is behaviour that is 3 standard deviations outside the normal. This is the sign of the free enterprise system, though. You can put a sign on your back saying "I want a heart operation", and 5 guys will jump out and do it no matter what it is for.

The additional problem with having invasive tests or surgery done to learn to fly is that the tools that are being used to select aviators now and in the future are going to become more and more complex. I think that we must spread the word among ophthalmologists who want to operate on young men who have not learned to fly, that they ought to satisfy themselves before they do a procedure on a young man, he must be qualified to fly on all other grounds. There is a certain tragedy in someone having his eyes operated on so that he can go to pilot training and he then shows up at an exam and he has prolapse and SVT. There are a myriad of problems that may cause disqualification from pilot training, and there is a certain tragedy about doing invasive studies or procedures only to find out that some of the other condiditions have not been met.

CASE PRESENTATIONS - TURKEY

Case 1

This 42 year old pilot presented in 1985 for his annual flight examination. He was asymptomatic, and the physical examination was normal. The EKG was normal except for left axis deviation. The total cholesterol was 292 mg/dl, and the total/HDL ratio was 6.6. He smoked about one pack per day of cigarettes. He had a family history of hypertension (mother), and his father had died from a myocardial infarction.

He was required to return in six months for re-evaluation because of his lipids.

One month later, he had an episode of chest pain and nausea, for which he did not seek medical attention, but treated himself with bedrest. He continued to have mild symptoms, but did not report them. Three weeks later, he developed angina during flight and had an episode of loss of consciousness in a dual pilot aircraft.

Again, his physical examinatiuon was unremarkable, with a blood pressure of 140/85. The EKG showed left axis deviation, and non-specific changes. On exercise testing, he developed 1 mm ST depression, with PVC's and then a 7 beat run of ventricular tachycardia. A repeat cholesterol was 282, HDL 42, and ratio 6.7. A thallium study showed a reversible perfusion defect, and cardiac catheterization showed single-vessel disease, with a 50% lesion in his left anterior descending coronary artery. He underwent percutaneous transluminal coronary angioplasty in the USA. He remains asymptomatic, with a normal examination and normal exercise ECG. He is on no medications.

Question: How often should we examine him and how? Can he be returned to flying?

Colonel Hickman: Since he had only 50% lesion in his LAD, you would have to worry about an element of coronary spasm contributing to this man's symptomatic angina. In the US Air Force, we do not return aviators to flying status after PCTA. I would not consider this man for flying duties without several Holters, a treadmill, thallium an a coronary catheterization. Since there has to be a question of spasm. I would want to treat him on clinical grounds with a calcium channel blocker, although this would d also be disqualifying in our Air Force.
There is also an ethical question of doing angioplasties in pilots to allow them to continue flying, although this was not the case here, where angiolplasty was indicated on medical grounds. The risk of the procedure, the significant risk of restenosis, which may be worse than the initial lesion, and the frequent follow-up which should include coronary angiography makes angioplasty an unattractive procedure for purely aeromedical reasons.

In aviators such as this man, with clearly a high risk of coronary artery disease, it would be worth doing a thallium study and fluroscopy. The issue is the dramatically increased risk, and the application of more tests to high risk patients rather than using your resources on low risk patients.

Case 2

This 52 year old General is a fighter pilot, with a past history of (duodenal) ulcer disease. He had recurring symptoms of abdominal pain, which was treated medically with ranitidine. He developed severe epigastric pain radiating to his chest and neck, and was taken to the Emergency Room, where an EKG showed a myocardial infarction. His previous EKG's and treadmills had all been normal including his most recent evaluation six months previously.

On coronary angiography he was found to have three vessel disease, and he subsequently underwent 3-vesal cornary artery bypass grafting. He has recovered well, with no further symptoms.

Question: Since he is a General, can we allow him to fly?

Colonel Hickman: In the US Air Force, General-rank aviators must retain their flying status in order to command. We recognize that they are where they are because of their special qualities, and that they are not there primarily to fly. We would therefore state that although aeromedically he is disqualified from flying, based on our medical assessment of the results and outcome of the CABG surgery, we feel he is safe to fly in non-high performance aircraft, basically as a passenger in his own aircraft.

Case 3

A 37 year old pilot developed severe chest pain and diaphoresis lasting several minutes just a few days before his annual examination. He reported this on this examination, and the EKG showed ST elevation and a Q wave in lead III. His only risk factor was cigarette smoking, one and a half packs per day. Because of the question of myocardial infarction, he underwent coronary angiography. The left ventriculogram was normal, but the coronary arteries were felt to show congential ectasia.

Discussion: Colonel Hickman and L/Colonel Kruyer felt there was ectasia of the left anterior descending and right coronary artery, and that there may have been a proximal LAD lesion as well, but without the films, were not willing to commit themselves. They recommended further investigation to include a treadmill, thallium and MUGA, and indicated that if there was no evidence of myocardial infarction, and no ischemia, and only ectasia, they would return the aviator to flying status.

Case 4

This 27 year old jet pilot's only apparent risk factor for coronary disease was cigarette smoking, one pack per day for 10 years. He developed severe crushing chest pain with nauseas and emesis, and at the hospital, was found to have an acute anterolateral myocardial infarction. He continues to have angina. The echo shows septal thinning and dyskinesis.

Discussion: Because of his age and continuing symptoms, Colonel Hickman and L/Colonel Kruyer recommended that he have coronary angiography, a thallium study and MUGA to assess the viable myocardium.

Everyone agreed that he is no longer fit for flying duties.

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